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CONGENITAL ABNORMALITIES IN INFANTS FOLLOWING INFECTIOUS DISEASES DURING PREGNANCY, WITH SPECIAL REFERENCE TO RUBELLA: A THIRD SERIES OF CASES.

By CHARLES SWAN[1] AND A. L. TOSTEVIN.

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THE ætiology of congenital malformations has been a subject of considerable conjecture. Although some observers had suggested that infection in the mother during pregnancy might be a factor, it was not until the publication of the work of Gregg[(1)] in 1941, that a definite relationship was established between the contraction of an infectious disease (rubella) in pregnancy and the occurrence of certain congenital defects (cataract and heart disease) in the infant born subsequently.

In South Australia, Swan, Tostevin, Moore, Mayo and Barham Black[(2)(3)] corroborated and amplified Gregg's findings. They concluded: (i) that detailed analysis of the symptomatology of the maternal disease failed to uphold any diagnosis other than that of German measles; (ii) that in addition to the abnormalities described by Gregg, there were other sequelæ, including deaf-mutism with or without cardiac disease, heart disease without any other defect, and microcephaly; (iii) that the "critical period" of pregnancy for the development of congenital abnormalities was the first three months; and (iv) that the pathogenesis of the malformations could best be interpreted on the basis of the known enhanced susceptibility of embryonic as compared with adult tissues to virus infections. (With regard to the association between congenital deaf-mutism and maternal rubella, we wish to state that although the relationship was first recorded by Swan and his associates,[(2)] recent correspondence[(4)(5)(6)] shows that the discovery was made independently by Gregg in New South Wales, and six months later by Tostevin in South Australia.)

Few accounts of the pathology of the congenital defects have been published. Gregg[(1)] referred briefly to the macroscopic autopsy findings in three subjects with cardiac lesions; in all the *ductus arteriosus* was widely patent; in addition, in one case patency of the *foramen ovale* and of the interventricular septum was found. Swan[(7)] described the pathological changes in three infants who died as a result of their malformations. In each instance the *ductus arteriosus* was widely patent; furthermore, in all cases variable degrees of patency of the *foramen ovale* were present and in one a defect of the interventricular septum was found. On microscopic examination the well-defined elastic lamina and intimal mounds normally present in the *ductus arteriosus* were absent; signs of obliterative endarteritis were non-existent. At the margins of the defect of the interventricular septum there was occasional replacement of muscle fibres by connective tissue. The lenses of the cataractous eyes were composed of a central "nuclear" portion, which appeared to have undergone necrosis *en masse*, and a cortical zone most evident in the equatorial region, consisting of degenerate lens fibres. A variable degree of glomerular sclerosis was observed in the kidneys. (The significance of the last-mentioned finding, however, remains to be determined[(8)].) Carruthers[(9)] examined the inner ears of an infant, aged six and a half months, who had congenital cataracts, heart disease and suspected deafness. The most conspicuous feature was ". . . the total absence, in both ears, of any differentiation of the primitive cells to form the organ of Corti".

[1] Work done with the aid of grants from the National Health and Medical Research Council, Australia, and the Keith Sheridan Research Fund of the University of Adelaide.

Evans[10] studied the congenital dental abnormalities. The main defect was retardation of eruption.

In their first paper Swan and his colleagues[2] concluded that the type of congenital defect was apparently determined by the stage of pregnancy at which the mother suffered from rubella. In an article read at the annual meeting of the Ophthalmological Society of Australia (British Medical Association) in October, 1944, Ida Mann[11] stated that in an attack by a noxious agent on an embryo, the organs affected were those in which the cells were in a state of active division. On this basis, using the data compiled by Swan and others,[2] she found that there was a definite correlation between the various types of abnormality observed and the period of pregnancy at which the mother contracted rubella.

In August, 1944, a committee which comprised Dr. N. McA. Gregg, Dr. D. Vickery, Dr. M. Heseltine, Dr. W. Ramsay Beavis, Dr. E. Meyers and Dr. A. E. Machin, was appointed by the Director-General of Public Health of New South Wales to investigate congenital malformations resulting from maternal rubella in pregnancy. The committee adopted the plan of investigation formulated by Swan *et alii*.[2] Most of the report[12] (based on 136 cases) consists of evidence confirming the original findings.[1,2,3,13] New observations include the following: (i) the fact that cases of congenital malformation subsequent to maternal rubella in pregnancy have occurred prior to the epidemics which started in 1939; (ii) the possibility that in rare instances the mother may contract German measles at an early stage of pregnancy and yet give birth to an apparently normal child; and (iii) the fact, foreshadowed by Carruthers,[9] that cataract and deaf-mutism (with or without heart disease) may coexist in the same patient.

Up to the present time detailed investigations have been carried out only in New South Wales and in South Australia. Cases are known to have occurred, however, in other Australian States, notably in Queensland[23] and Western Australia.[14] In the last-mentioned State a nursery school for the treatment of deaf-mutism has been opened.

Other contributions to the Australian literature include reviews with additional data by Gregg[13,12] and by Swan.[5]

Recently investigators overseas have begun to record cases. In America reports include those of Reese[17] (three cases), Rones[18] (three cases), Erickson[19] (11 cases), Perera[20] (one case), Greenthal[21] (two cases) and Altmann and Dingmann[22] (one case). In England examples have been observed by Hope Simpson[23] (two cases), Martin[24] (24 cases) and Hughes[25] (one case). One case has been observed in New Zealand.[26] In all instances these results confirm the Australian work.

In the present paper we record the results of study of a third series of cases of congenital malformation associated with infectious disease in the mother during pregnancy. As far as rubella is concerned, we believe that we have now collected the vast majority of cases in which congenital defects were present, which have occurred in South Australia since 1939.

The plan of the investigation was that formulated earlier.[2,12] One of us (C.S.) collected and correlated the data and carried out the pathological and the routine physical examinations; the other (A.L.T.) examined the eyes and ears of the children.

Results of the Investigation.

The series comprised 58 cases (Table I). It consisted of 40 cases of rubella in pregnancy and two cases in which German measles was contracted less than a fortnight prior to conception. In addition there were 16 cases in which the mother had suffered from other infectious diseases during pregnancy, including morbilli (eight cases), mumps (three cases), chicken-pox (two cases), *herpes zoster* (two cases) and scarlet fever (one case).

A few of the cases have been briefly referred to in a recent review.[3] The numbers adopted are a continuation of those used previously.[2,12] (Case 105 was designated Case "Z" in an earlier paper.[12])

Rubella During Pregnancy and Congenital Defects.

Geographical Distribution.

At the time when they contracted rubella, 35 of the mothers lived in South Australia, 24 of them in the metropolitan area of Adelaide and 11 in the country districts. Of the remainder, two resided in New South Wales, one in Victoria, one in Western Australia and one in Scotland.

Annual Incidence of Cases.

In one case the exanthem occurred in 1938, in seven cases it occurred in 1939, in 12 in 1940, in five in 1941, in seven in 1942, in four in 1943 and in three in 1944. In the remaining case the infection was contracted either in 1940 or in 1941.

Nature and Manifestations of the Exanthematous Disease.

In order to determine more precisely the character of the disease, one of us (C.S.) interrogated all the mothers. Although with the passage of time in a few instances the details had been forgotten, usually it was possible to obtain a reasonably satisfactory account of the illness.

Of the 40 cases, in 22 (Cases 76, 77, 79, 84, 86, 89, 90, 91, 92, 93, 94, 95, 96, 97, 99, 100, 102, 104, 105, 107, 108, and 114) the diagnosis of German measles was made by medical practitioners. In Case 106 the exanthem was diagnosed as a "nervous" rash, but questioning revealed that at the time the patient was frequently in contact with a niece suffering from "measles". In Cases 98 and 103 the husband, and in Case 92 a nephew, had suffered from rubella (diagnosed by medical practitioners) about a fortnight before. In Case 80 a diagnosis of German measles was made by a chemist; at about the same time three members of the household and a friend also contracted the disease. In Cases 87, 101 and 109 there was an epidemic of rubella in the district at the time (in the first two instances other persons in the household also had the infection). In Case 81 other members of the family had suffered from German measles a few weeks before the patient contracted the disease.

Premonitory Symptoms.

Premonitory symptoms were present in 22 instances (Cases 75, 76, 78, 79, 80, 81, 83, 84, 85, 87, 89, 91, 93, 94, 96, 98, 99, 102, 103, 104, 108, and 109). They preceded the rash by intervals which ranged from one to seven days and averaged three days.

The commonest symptom was tiredness or sleepiness, which occurred in 12 cases (Cases 76, 78, 79, 81, 83, 89, 91, 99, 102, 104, 108 and 109). Nine mothers (Cases 76, 78, 79, 81, 84, 85, 87, 96 and 102) stated that they were "off-colour", three (Cases 80, 85 and 93) complained of a feeling of "heaviness", and three (Cases 84, 85 and 93) felt as though they were getting a "cold" or influenza. Seven women (Cases 76, 79, 87, 89, 96, 99 and 108) suffered from headache, six (Cases 80, 89, 91, 93, 102 and 103) from generalized aching, and two (Cases 104 and 109) from backache. Vomiting occurred in four instances (Cases 80, 84, 89 and 98), nausea in two (Cases 79 and 99), and anorexia in three (Cases 78, 79 and 85). Lymphadenitis (post-auricular and/or cervical) was present in three cases (Cases 91, 94 and 108), photophobia in two (Cases 79 and 93) and sore throat in two (Cases 78 and 102). Shivering attacks were present in two cases (Cases 75 and 83). Other symptoms included "cold in the head" (Case 78), vertigo (Case 79), mental depression (Case 80), irritability (Case 104) and fainting (Case 80).

Character of the Exanthem.

The typical rash was composed of small spots, about the size of pins' heads, red or pink in colour and packed closely together. In nine instances (Cases 77, 79, 81, 82, 87, 96, 107, 108 and 113), however, the spots were considered to measure as much as twice the size of pins' heads. The rash in Cases 77 and 81 was described as "blotchy", while in Case 113 it resembled "mosquito bites". In Case 106 the usual rash was combined with "red blotches all over the skin". In Case 93 the rash was typical on the body and "blotchy on the face". Two patients (Cases 75 and 104) stated that "the spots ran together".

TABLE I.

Analysis of Type and Period of Onset of Infectious Disease during Pregnancy and of the Congenital Defects in the Infants Born Subsequently.

Case Number.	Place of Residence of Mother at Time of Disease.[1]	Age in Years.	Disease During Pregnancy.	Number of Months Pregnant at Onset of Disease.	Date of Last Menstrual Period Prior to Pregnancy.	Date of Birth of Baby.	Expected Date of Birth.	Sex of Child.	Birth Weight. (Pounds and Ounces.)	Circumference of Skull (Inches); Normal Measurement for Age and Sex in Parentheses.	Congenital Defects.	Remarks.
75	Aberdeen, Scotland.	38	Rubella.	Approximately 0·25	20. 9.38	27. 6.39	27. 6.39	M.	6, 10	20·5 (20·0)	Heart disease, pyloric stenosis.	
76	Croydon Park.	32	Rubella, possibly mumps also.	0·5 to 0·75	15.12.42	20. 9.43	22. 9.43	F.		18·3 (18·2)	Cleft palate (soft).	
77	Colonel Light Gardens.	—	Rubella.	0·5 to 1·0	Early in August, 1942.	27. 5.43	—	M.	5, 0	17·0 (18·6)	Heart disease.	
78	Kangaroo Island.	21	Rubella.	Approximately 1·0	October, 1940.	19. 6.41	—	F.	2, 4	17·8 (19·5)	Deaf - mutism, heart disease.	Approximately one month premature.
79	Woodlands Park.	30	Rubella.	1·25[2]	1. 9.41	13. 5.42	8. 6.42	M.	5, 13·5	18·9 (19·8)	Deaf - mutism, heart disease.	
80	Woodville Park.	28	Rubella.	1·5[2]	Approximately 12. 9.39	30. 5.40	Approximately 19. 6.40	F.	5, 0	18·4 (19·6)	Deaf - mutism, heart disease.	
81	Sydney, New South Wales.	35	Rubella.	1·5	Approximately 22. 6. 40	30. 3.41	Approximately 29. 3.41	M.	7, 0	19·5 (19·8)	Deaf - mutism, heart disease.	
82	Nuriootpa.	21	Rubella.	1·5	Unknown.	9. 6.40	Unknown.	F.	5, 6	18·8 (19·5)	Deaf-mutism.	Two weeks premature.
83	North Adelaide.	24	Rubella.	Approximately 1·5	Late in June, 1940.	16. 4.41	—	F.	6, 9·25	19·3 (19·4)	Deaf-mutism.	
84	Rosewater Gardens.	19	Rubella.	Approximately 1·5	End of July, 1942.	16. 4.43	—	M.	—	18·5 (19·3)	Deaf - mutism, heart disease.	
85	Mildura, Victoria.	21	Rubella.	Approximately 1·5	10. 5.43	26. 2.44	17. 2.44	F.	5, 0	13·8 (14·5)	Unilateral cataract, deaf-mutism, heart disease.	
86	Broken Hill, New South Wales.	20	Rubella.	Approximately 1·5	Unknown.	25. 3.40	Unknown.	M.	6, 1	18·5 (20·0)	Deaf-mutism.	Approximately three weeks premature.
87	Rowland's Flat.	21	Rubella.	Approximately 1·5	Unknown.	1. 7.40	Unknown.	M.	5, 10	19·4 (19·9)	Deaf-mutism, nævus in occipital region.	
88	Stepney.	23	Rubella.	1·0 to 2·0	Approximately 17.11.40	5. 9.41	Approximately 24. 8.41	M.	4, 15·5	18·1 (19·9)	Deaf-mutism.	
89	Goodwood.	33	Rubella.	1·75[2]	18.12.39	25. 9.40	25. 9.40	M.	6, 12	18·6 (19·9)	Deaf - mutism, heart disease.	
90	North Adelaide.	24	Rubella.	1·5 to 2·0	End of June, 1942.	6. 4.43	—	F.	6, 0	18·5 (18·6)	Deaf-mutism.	
91	Pinery.	30	Rubella.	1·75 to 2·0	Approximately 26. 6.43	—	—	M.	—	—	Unilateral cataract, with lack of closure of the fœtal (choroidal) fissure.	Mother aborted when 2·5 months pregnant.
92	Burra.	27	Rubella.	2·0[2]	25.10.39	24. 7.40	1. 8.40	M.	6, 4	19·5 (20·0)	Deaf-mutism.	
93	Knoxville.	24	Rubella.	2·0[2]	26. 5.44	18. 3.45	2. 3.45	F.	7, 9·5	15·6 (14·0)		
94	Colonel Light Gardens.	21	Rubella.	2·0[2]	14.6.40	3. 3.41	21. 3.41	M.	8, 0	19·8 (19·9)	Deaf-mutism.	
95	Marryatville.	—	Rubella.	2·0[2]	30.10.42	22. 7.43	6. 8.43	M.	6, 6	18·3 (18·7)	Microcephaly.	

[1] Unless otherwise stated, in State of South Australia.
[2] Duration of pregnancy calculated from fourteenth day after onset of the last menstrual period.

TABLE I.—*Continued.*

Analysis of Type and Period of Onset of Infectious Disease during Pregnancy and of the Congenital Defects in the Infants Born Subsequently.—Continued.

Case Number.	Place of Residence of Mother at Time of Disease.[1]	Age in Years.	Disease During Pregnancy.	Number of Months Pregnant at Onset of Disease.	Date of Last Menstrual Period Prior to Pregnancy.	Date of Birth of Baby.	Expected Date of Birth.	Sex of Child.	Birth Weight. (Pounds and Ounces.)	Circumference of Skull (Inches); Normal Measurement for Age and Sex in Parentheses.	Congenital Defects.	Remarks.
96	Parkside.	27	Rubella.	2·0[2]	11.4.42	31.12.42	18. 1.43	M.	6, 0	17·9 (15·4)	Deaf - mutism, heart disease, left concomitant strabismus.	
97	Semaphore.	22	Rubella.	2·0	July, 1941.	19. 4.42	—	F.	6, 6	17·9 (19·3)	Deaf-mutism.	
98	Tusmore.	27	Rubella.	Approximately 2·0	10.10.39	31. 7.40	17. 7.40	M.	6, 13	19·6 (19·9)	Deaf-mutism.	
99	Claremont, Western Australia.	25	Rubella; soldier's throat.	2·25[2] early in pregnancy.	2. 7.40	22. 4.41	9. 4.41	M.	6, 5	20·0 (19·9)	Deaf - mutism, heart disease.	
100	Tanunda.	34	Rubella.	2·25[2]	3.11.41	16. 8.42	10. 8.42	M.	5, 14	18·8 (19·3)	Deaf-mutism.	
101	Tintinara.	31	Rubella.	2·0 to 3·0	Unknown.	25. 6.41	Unknown.	M.	6, 9	19·3 (19·8)	Deaf - mutism, heart disease.	
102	North Adelaide.	—	Rubella.	2·75[2]	1. 5.42	7. 2.43	8. 2.43	M.	6, 6	18·6 (19·2)	Deaf - mutism, heart disease.	
103	Lobethal.	25	Rubella.	3·0	9. 6.41	3. 3.42	16. 3.42	M.	7, 14·5	20·0 (19·9)	Deaf - mutism, heart disease.	
104	Nairne.	31	Rubella.	3·0	Unknown.	21. 8.41	Unknown.	M.	8, 2	19·9 (19·5)	Deaf - mutism, heart disease.	
105	Semaphore.	29	Rubella.	Approximately 3·0	8. 6.42	15. 2.43	15. 3.43	F.	5, 8	17·5 (18·0)	Microcephaly, backwardness in development.	
106	Port Pirie.	19	Rubella.	Approximately 3·0	Unknown.	26. 2.40	Unknown.	F.	6, 0	19·1 (19·5)	Deaf-mutism.	
107	Rose Park.	18	Rubella.	3·5[2]	28. 5.44	24. 2.45	4. 3.45	M.	7, 13	13·9 (14·0)		
108	College Park.	26	Rubella.	3·5[2]	4. 9.43	6. 6.44	11. 6.44	M.	7, 8	14·6 (14·7)	Heart disease.	
109	Woodside.	—	Rubella.	4·0	Unknown.	17. 5.40	Unknown.	F.	7, 0	20·0 (19·6)	Deafness, heart disease.	
110	Adelaide.	22	Rubella.	4·5[2]	18. 5.40	5. 3.41	25. 2.41	M.	5, 4	19·8 (19·9)	Speech defect, heart disease.	
111	Westbourne Park.	28	Rubella.	6·0	Unknown.	24. 9.40	Unknown.	M.	7, 11	20·3 (20·0)		
112	Albert Park.	29	Rubella.	6·0	7. 6.41	23. 3.42	14. 3.42	M.	7, 6	20·1 (19·9)	*Spina bifida occulta.*	
113	Woodville.	31	Rubella.	7·0 to 8·0	Unknown.	3.11.40	Unknown.	M.	6, 0	19·1 (19·8)	Mongolism, heart disease.	
114	Colonel Light Gardens.	—	Rubella.	Indeterminate.	9. 8.40	31. 3.41	16. 5.41	M.	5, 11	20·1 (19·9)	Spastic diplegia, pyloric stenosis, right inguinal hernia, strabismus.	Thought to be six weeks premature.
115	Glen Osmond.	26	Rubella.	Approximately 13 days before onset of pregnancy.	13. 2.45	—	—	M.	—	—		Mother aborted 14.6.45.

[1] Unless otherwise stated, in State of South Australia.
[2] Duration of pregnancy calculated from fourteenth day after onset of the last menstrual period.

TABLE I.—*Continued.*

Analysis of Type and Period of Onset of Infectious Disease during Pregnancy and of the Congenital Defects in the Infants Born Subsequently.—Continued.

Case Number.	Place of Residence of Mother at Time of Disease.[1]	Age in Years.	Disease During Pregnancy.	Number of Months Pregnant at Onset of Disease.	Date of Last Menstrual Period Prior to Pregnancy.	Date of Birth of Baby.	Expected Date of Birth.	Sex of Child.	Birth Weight. (Pounds and Ounces.)	Circumference of Skull (Inches); Normal Measurement for Age and Sex in Parentheses.	Congenital Defects.	Remarks.
116	Prospect.	28	Rubella.	Eight days after start of last menstrual period, approximately six days before onset of pregnancy.	4.11.43	14. 8.44	11. 8.44	F.	5, 0	15·6 (15·0)		
117	Renmark.	35	Morbilli.	0·5	12. 9.44	28. 6.45	19. 6.45	F.	7, 9·5			
118	South Plympton.	29	Morbilli.	2·0	1. 3.44	13. 1.45	8.12.44	F.	8, 5	13·9 (13·5)		
119	Prospect.	27	Morbilli.	2·5	Approximately 2. 5.44	16. 2.45	Approximately 9. 2.45	F.	—	14·8 (14·4)		
120	Hindmarsh.	25	Morbilli.	3·0	28. 6.44	17. 4.45	5. 4.45	F.	8, 0	14·4 (13·6)		
121	Nuriootpa.	34	Morbilli.	3·5	13. 6.44	22. 3.45	20. 3.45	M.	6, 12	17·1 (15·9)	Heart disease, pyloric stenosis.	
122	Goodwood.	22	Morbilli.	3·75[2]	2. 6.44	2. 3.45	9. 3.45	M.	6, 11	15·0 (14·5)		
123	Aldgate.	—	Morbilli.	4·0	18. 5.42	27. 2.43	25. 2.43	M.	—	19·8 (19·2)	*Genu valgum.*	
124	Queenstown.	30	Morbilli.	8·0	7.11.43	16. 8.44	14. 8.44	M.	8, 12	16·5 (15·7)		
125	Norwood.	29	Mumps.	0·25[2]	19.10.44	28. 7.45	26. 7.45	F.	7, 5·5	14·6 (14·1)	Slight capillary nævus, right upper eyelid.	Mother also has bronchiectasis.
126	Da Costa Park.	33	Mumps.	Approximately 0·75	6. 9.43	26. 6.44	13. 6.44	F.	8, 6	18·0 (17·4)	Imperforate anus.	
127	North Adelaide.	27	Mumps.	7·0	12. 5.44	20. 2.45	19. 2.45	M.	8, 4	13·6 (14·0)	Microcephaly.	
128	Norwood.	27	Varicella.	6·0 to 7·0	2. 5.44	18.2.45	9. 2.45	F.	8, 1·5	16·6 (16·6)	Heart disease, nævus of scalp.	Mother also had "boils in ears" when five months pregnant.
129	Adelaide.	—	Varicella.	7·0	23. 3.44	11. 1.45	30.12.44	F.	7, 10	14·8 (14·5)		
130	Torrens Park.	23	*Herpes zoster.*	4·0	Unknown.	18. 4.41	Unknown.	F.	6, 1	18·8 (19·5)	Deaf - mutism, heart disease.	
131	North Adelaide.	20	*Herpes zoster.*	6·0	5. 9.44	16. 6.45	12. 6.45	F.	7, 2	14·3 (13·5)	Myelocele, bilateral talipes, right *genu recurvatum.*	
132	Colonel Light Gardens.	22	Scarlet fever.	7·0	23. 9.44	14. 7.45	30.6.45	M.	5, 9	14·0 (14·0)	Obliteration of bile ducts.	Child died.

[1] Unless otherwise stated, in State of South Australia.
[2] Duration of pregnancy calculated from fourteenth day after onset of the last menstrual period.

Itchiness was variable; in 16 instances the rash was itchy and in 14 it was the reverse.

Fourteen mothers observed that the spots were raised and seven that they were not raised; in the remainder no information was available.

In Case 75 subsequently the skin peeled.

Distribution of the Rash.

For the most part, the eruption commenced on the face, arms or chest (less often on the abdomen) and spread over the entire body. In Cases 76, 82, 94, 101, 102 and 105 the exanthem was generalized from the outset. In Case 75 the rash was believed to have been limited to the chest, in Case 86 to the trunk, in Cases 77 and 113 to the face and chest, in Case 81 to the face and neck, in Case 98 to the face and trunk, in Case 80 to the neck and shoulders, in Case 99 to the arms and chest, in Case 84 to the arms and trunk, in Case 88 to the legs, trunk and face, and in Cases 100 and 112 to the lower limbs and abdomen.

Duration of the Exanthem.

Usually the rash lasted for three days. In Cases 95 and 103 the mothers stated that the eruption was present for five days, in Case 107 for about six days, in Cases 77, 91, 92 and 110 for a week and in Case 89 for seven to 10 days.

Lymphadenitis.

Enlargement of the post-auricular and/or cervical lymph glands to the size of peas or beans was present in 18 instances (Cases 76, 77, 79, 85, 89, 90, 91, 93, 94, 95, 97, 102, 103, 105, 108, 109, 110 and 111), absent in seven instances (Cases 81, 82, 96, 99, 101, 104 and 112) and doubtful or "not known" in the remainder. In Case 76 the swelling of the left post-auricular and cervical glands was so pronounced that the patient was suspected by her medical attendant to be suffering from mumps as well.

Concomitant Signs and Symptoms.

Photophobia and soreness of the eyes were complained of in 14 cases (Cases 76, 78, 79, 80, 81, 85, 90, 91, 93, 96, 107, 108, 110 and 113) and denied in 21 cases (Cases 77, 82, 83, 84, 86, 89, 92, 94, 95, 97, 99, 100, 101, 102, 104, 105, 106, 109, 111, 112 and 114).

Cough was absent in 27 cases, and doubtful or not remembered in seven cases (Cases 87, 90, 97, 98, 100, 104 and 111). Of the remainder, four patients (Cases 75, 79, 84 and 86) had slight coughs, and two (Cases 93 and 94) had severe hacking coughs lasting two or three weeks.

Arthritis was experienced in eight cases; it was characterized by pain and stiffness in the joints, intensified on movement. In Case 75 it affected most of the joints, in Case 77 the neck, knees and wrists, in Case 79 the fingers, in Case 86 the knees, in Case 89 the elbows and knees, in Cases 100 and 103 the wrists, and in Case 108 the knees, elbows and wrists. In addition, in Case 91 the patient stated that her hands and knees were swollen, but that no stiffness or aching was observed. The mother in Case 92 complained of aching of her arms.

Malaise occurred in five cases (Cases 79, 90, 92, 110 and 113), headache in three (Cases 78, 80 and 108) and giddiness in two (Cases 83 and 101).

Three patients (Cases 83, 95, and 105) stated that they felt "hot and burning", and two (Cases 80 and 89) suffered from stuffiness of the nose.

Two mothers (Cases 83 and 104) complained of tiredness; the latter said that she felt extremely tired "for weeks afterwards".

Infrequent symptoms included vomiting (Case 77), anorexia (Case 83), thirst (Case 110), sore throat (Case 101), and earache (Case 80). In Case 96 the patient noted spots under her nails for about four days.

Source of Infection.

The probable sources of infection were considered to be as follows: In Cases 78, 96, 98, 100 and 103 the husband, in Case 91 a brother, in Case 92 a nephew, in Case 106 a niece, and in Cases 80 and 87 other members of the household.

Transmission to Others.

Four mothers (Cases 90, 92, 97 and 103) each believed that they had infected a member of their family or of the household.

Previous Infections.

If two doubtful cases (Cases 93 and 113) are omitted, the rest of the patients with the exception of seven (Cases 77, 78, 86, 87, 106, 109 and 110) had suffered from morbilli. Twenty mothers (Cases 75, 78, 79, 81, 82, 83, 84, 87, 92, 95, 96, 98, 99, 100, 103, 107, 108, 109, 111 and 112) had had mumps, thirteen (Cases 77, 79, 82, 84, 85, 90, 93, 95, 96, 97, 101, 104 and 108) had had varicella, 21 (Cases 77, 79, 80, 82, 83, 85, 88, 90, 95, 96, 97, 98, 99, 100, 101, 102, 103, 104, 108, 111 and 114) had had whooping cough, four (Cases 80, 87, 91 and 96) had had diphtheria, and seven (Cases 75, 83, 93, 95, 98, 102 and 109) had had scarlet fever. It is noteworthy that in only five instances (Cases 76, 85, 90, 104 and 112) was there a previous history of rubella; in two of these (Cases 76 and 85) the history was rather vague.

Miscarriages.

Four mothers had had a miscarriage, three of them (Cases 80, 91 and 93) prior to and one (Case 87) subsequent to the pregnancy in which they had contracted German measles.

Babies Resulting from Pregnancies Subsequent to that in which the Mother Suffered from Rubella.

Eleven mothers (Cases 78, 80, 83, 84, 91, 98, 99, 103, 106, 111 and 114) had given birth to a further child, and three (Cases 86, 87 and 92) to two further children. In all instances the children were normal. The fact that a mother has given birth to a congenitally defective child as the result of rubella in pregnancy does not mean, therefore, that further pregnancies are contraindicated.

Relation of the Period of Pregnancy at which the Exanthem Occurred to the Development of Congenital Abnormalities in the Child.

In certain instances (Cases 79, 80, 89, 92, 93, 94, 95, 96, 99, 100, 102, 107, 108 and 110) (marked * in Table I) knowledge of the exact dates of the last menstrual period and of the onset of the rash permitted more accurate estimation of the stage of pregnancy at which the infection occurred. Conception was presumed to have taken place fourteen days from the first day of the last menstrual period.

Of the 37 mothers who later gave birth to congenitally defective offspring, 31 had contracted rubella in the first three months of pregnancy (Table II). Four had suffered from the disease in the first month of pregnancy, 19 in the

TABLE II.

Relationship between the Time of Contraction of Rubella during Pregnancy and the Occurrence of Congenital Malformations in the Infants Born Subsequently.

Month of Pregnancy.	Number of Infants with Congenital Defects.	Number of Normal Children.	Total.
0 to 1	4	—	4
1 to 2	19	1	20
2 to 3	8	—	8
3 to 4	2	1	3
4 to 5	1	—	1
5 to 6	1	1	2
6 to 7	—	—	0
7 to 8	1	—	1
8 to 9	—	—	0
Indeterminate	1	—	1
Total	37	3	40

second month and eight in the third month. Of the six exceptions, two (Cases 108 and 109) had contracted the infection in the fourth month, one (Case 110) in the fifth month, one (Case 112) in the sixth month, and one (Case 113) in the eighth month; in Case 114 the precise period of pregnancy at which the mother had suffered from German measles could not be ascertained.

In three cases in which the babies born subsequently were apparently normal, one mother (Case 93) had had rubella in the second month of pregnancy, one (Case 107) in the fourth month and one (Case 111) in the sixth month.

The Nature of the Congenital Defects.

Of the 37 babies with defects 26 were males and the rest females.

The average birth-weight of 34 of the infants with congenital malformations was six pounds two and a half ounces. One baby weighed between two and three pounds, one weighed between four and five pounds, 10 weighed between five and six pounds, 15 weighed between six and seven pounds, five weighed between seven and eight pounds, and two weighed between eight and nine pounds. The respective birth-weights of the three apparently normal infants were seven pounds nine and a half ounces, seven pounds 13 ounces and seven pounds 11 ounces.

In more than 20 instances it was possible to compare the weight and height of the child with the standard for age and sex. The children in the following 15 cases were underweight; Case 78 (10 pounds underweight), Case 77 (six and a half pounds), Cases 79, 86 and 97 (each five pounds), Case 92 (four and a half pounds), Cases 80, 88 and 89 (each four pounds) and Cases 83, 84, 87, 90, 101 and 114 (three pounds or less). In Cases 82, 99 and 108 the weight was normal. The children in the following seven cases were overweight: Case 75 (11 pounds overweight), Case 112 (seven pounds), Cases 94, 109 and 110 (each five pounds) and Cases 98 and 103 (three pounds or less). The children in the following eight cases were below standard height: Case 80 (three and a half inches less than normal), Cases 83 and 86 (two to three inches) and Cases 79, 84, 88, 92 and 114 (one to two inches). Six children were taller than normal (Case 94, three inches above standard height; Cases 98, 103 and 112, approximately two inches; Cases 90 and 110, one inch); variations of half an inch or less were disregarded.

When variations of less than a week between the actual and expected dates of birth are set aside, 13 infants (Cases 78, 79, 80, 82, 86, 92, 94, 95, 96, 103, 105, 107 and 114) were premature. In three instances (Cases 79, 105 and 114) they were a month or more premature. All except one of these babies (Case 107) were congenitally defective.

With regard to the position of the infants in their respective families, 18 (Cases 80, 82, 83, 84, 85, 86, 88, 92, 94, 97, 98, 99, 102, 103, 108, 110, 112 and 114) were the first children, 11 (Cases 75, 76, 78, 87, 90, 96, 100, 104, 105, 106 and 109) were the second children, four (Cases 77, 79, 95 and 113) were the third children, one (Case 89) was the fifth child and one (Case 101) was the sixth child.

Sixteen mothers (Cases 76, 77, 78, 84, 88, 92, 94, 96, 98, 100, 101, 102, 105, 110, 113 and 114) complained that their babies were difficult to feed.

Eye Defects.

There were four subjects (three infants and a fœtus) with eye defects (Table III). In three instances (Cases 85, 91 and 96) the mothers were in the second month of pregnancy when they contracted rubella; in the remaining instance (Case 114) the duration of pregnancy at the onset of the exanthem was not determined. Two of the cases (Cases 85 and 91) had unilateral cataract (right eye). In the former case the baby suffered also from deaf-mutism and heart disease. In its general character and in the associated microphthalmos and difficulty in mydriasis, the cataract in Case 85 was similar to that described by Gregg;[1] it also showed the prominence of the Y sutures, originally emphasized in an earlier paper[2] by Barham Black. In Case 91 the cataract was accompanied by lack of closure of the fœtal (chorioidal) fissure (for illustrations see Swan[3]).[1] In this connexion it is of interest that in one of Gregg's[1] cases of monocular cataract examination of the non-cataractous eye ". . . under mydriasis revealed a large, pale area with some scattered pigmentation in the lower half of the fundus suggestive of a coloboma". In the remaining two cases (Cases 96 and 114) concomitant strabismus was present. In the former case it was accompanied by deaf-mutism and heart disease, in the latter by spastic diplegia, pyloric stenosis and inguinal hernia. The association of this abnormality with German measles may quite well be purely fortuitous; its incidence would not appear to be above the average of that in otherwise apparently normal children. In this regard the New South Wales committee has reached a similar conclusion.[12]

Deaf-Mutism and Speech Defects.

The series of cases of deaf-mutism and speech defects comprised 27 patients (18 males, nine females); it included 22 certain cases of deaf-mutism, four (Cases 80, 96, 98 and 109) in which the degree of deafness was slight or doubtful, and one (Case 110) of speech-defect (Table IV). Fifteen children (Cases 78, 79, 80, 81, 84, 85, 89, 96, 99, 101, 102, 103, 104, 109 and 110) suffered also from heart disease. In Case 85 there was also unilateral cataract, in Case 87 a nævus, and in Case 96 strabismus. In one instance the mother had had German measles probably in the first month of pregnancy, in 17 instances in the second month, in seven

[1] On page 135, line 20, of the article,[3] the age of the fœtus is described erroneously as three instead of two and a half months.

TABLE III.

Analysis of Congenital Eye Defects following Rubella during Pregnancy.

Case Number.	Nature of Eye Defects.	Associated Defects.	Treatment.	Remarks.
85	Right-sided cataract. The mother noticed a "film" on the pupil of the eye when the baby was four weeks old. Examination revealed also right microphthalmos. The right pupil dilated poorly with the mydriatic. The Y-sutures of the cataractous lens were prominently marked. The media of the left eye were clear and the *fundus oculi* was normal.	Heart disease, deaf-mutism.	—	—
91	Considerable disorganization of structure of the right eye associated with microphthalmos, cataract and lack of closure of the fœtal (chorioidal) fissure.	—	—	Mother aborted when about two and a half months pregnant.
96	Left concomitant strabismus. Mother does not know whether the condition was present at birth, but it was certainly evident at the age of three weeks, and became worse after an attack of bronchitis at the age of four months. On examination the lenses of both eyes appeared to be normal.	Deaf-mutism, heart disease.	The "good" eye was covered for nine months, but in spite of this treatment there was no response—that is, fixing on the part of the left eye.	Mother had intermittent squint until the age of twelve years, but the condition righted itself without treatment.
114	Intermittent concomitant strabismus.	Spastic diplegia, pyloric stenosis, right inguinal hernia.	—	—

TABLE IV.

Analysis of Congenital Deaf-mutism and Speech-defects following Rubella in Pregnancy.

Case Number.	Age at Time of Examination. (Years and Months.)	Hearing.	Speech.	Hearing Tests with Tuning Forks of High (2,048) and Low (512) Pitches.	Associated Defects.
78	3, 11	Mother believes that child is almost "stone deaf". May hear a tray being dropped, but not motor-cars, aeroplanes, telephones, or alarm clocks.	Limited to "mummy" and "bub"; vocalizes.	No apparent response.	Heart disease.
79	3, 1	Deafness first suspected at age of 18 months. Can hear motor-cars, a train passing, train whistles, aeroplanes, the wireless, and children laughing and crying. Does not respond to requests, and sometimes appears puzzled as to what is required of him.	First spoke at age of 14 months, but made no further progress until two to three months before examination. Can now say "mum", "dad", "Louise", "look", "no" and "there you are". Sings loudly, but not in tune. Calls out loudly.	Difficult to assess owing to lack of cooperation.	Heart disease.
80	5	As a baby appeared to hear satisfactorily, and at the age of one year said "dad", "mum" and "bubba". At the age of 20 months for about a week had giddy spells in which she would "go white and vacant-looking and would put her head on the ground". After this attack her speech did not progress. At 3·5 years had an antrostomy, tonsillectomy and adenoidectomy and then started to speak. Now hears common sounds but not always if a long distance away. Hears wireless, telephone, baby crying, motor-cars and aeroplanes.	Now says single words and sentences of up to six words. Will name common objects provided she has previously been told their names. Can count up to five. Speech is imperfect—for "yellow" says "innow", for "blue" says "moo", for "black" says "mack", and for "grey" says "way".	Hears high and low tuning forks moderately well, and equally in the two ears. Deaf-mute's type of voice.	Heart disease.
81	3, 4	Mother noted that the child did not cry normally—"had a sort of squeal". He was first considered to be deaf at 2·75 years. Can hear a whistle, loud wireless, hand clapping and a telephone ringing. Sometimes he hears aeroplanes.	Can say "ta" and "ah".		Heart disease.
82	4, 10	Can hear her name, "Rosemary", if said loudly. Also hears the ticking of a clock, a church bell, the telephone ringing, train whistles, cows bellowing, aeroplane engines and motor-car horns.	Up to the age of 2·5 years made no attempt to speak. Then was able to say "pussy" and "dad". At three years was able to say also "Peter", "moo-cow" and "chick-chick". Says "choo-choo" in imitation of a train. Has never been able to put sentences together. Can count up to four.	No apparent response.	
83	4, 4	Deafness first suspected at 1·5 years. Does not hear people walking up to her. Appears to hear aeroplanes, the wireless, whistling and hand-clapping. Hears high-pitched sounds. Will imitate a "tommy" gun. Can hear through a telephone receiver and ear-phones.	First spoke at age of 2·5 years. Says "bubba", "ta", "Pat" and "for". Says "un" for "one".		
84	2, 2	Does not seem to hear anything. Does not hear aeroplanes, motor-car horns, hand clapping or people approaching him.	Limited to "mum". Vocalizes.	No apparent response.	Heart disease.
85	1, 9	Deafness first suspected at age of one year. Child does not respond to calling. Does not hear a dog barking, the telephone ringing, or alarm clocks. Imitates a woman imitating an aeroplane engine.	Vocalizes. Growls. Makes no attempt to speak.	No apparent response.	Unilateral cataract, heart disease.
86	5, 2	May perhaps hear wireless when turned up very loudly; this may, however, be due to the vibration. Otherwise appears to be "stone deaf".	Limited to "mum" and "bub".	No apparent response.	
87	4, 9	Deafness first suspected at age of one year. Would not respond to calling. Hears aeroplanes, trains, the telephone ringing and perhaps the loud high-pitched screaming of other children.	For the last two years has said "mum"; before that he said nothing. Makes noises and squeals.	No apparent response.	Nævus in occipital region.
88	3, 10	Deafness first suspected at 18 months when the child did not speak. He can now hear his father whistling, loud wireless, aeroplanes (when flying low), train whistles, hissing of steam-engines, and sometimes people walking up to him. He tends to hear high-pitched rather than low-pitched sounds. Probably he does not hear the telephone ringing. He hears the dog barking and tries to imitate it.	Says "mum". Vocalizes.	Hears high tones with right ear and not with left. No response to low tones in either ear.	
89	4, 6	Deafness was first suspected at eight months. The mother could drop things and make noises near the child without his noticing. He can now hear the telephone ringing, a baby crying, bells ringing, children shouting, aeroplanes, train whistles, the wireless and screaming. He appears to hear better with the right ear than the left; if he wants to concentrate on a sound he flexes his head and closes his eyes.	Up to the age of four years made no attempt to speak. Made a peculiar type of screech; was able to laugh heartily. After two months' speech therapy was able to say "mum", "dad", "ta", "ta-ta", "fire" and "kitty" (for "cat").	No apparent response.	Heart disease.
90	2	Does not hear her mother walking up behind her on the gravel. Does not respond to her name. Can hear hand clapping, the telephone, wireless and a motor-car horn.	Limited to "mum" and "ta".		

TABLE IV.—*Continued.*

Analysis of Congenital Deaf-mutism and Speech-defects following Rubella in Pregnancy.—Continued.

Case Number.	Age at Time of Examination. (Years and Months.)	Hearing.	Speech.	Hearing Tests with Tuning Forks of High (2,048) and Low (512) Pitches.	Associated Defects.
92	5, 1	Deafness first suspected at 18 months. Does not hear people approaching him. Hears aeroplanes, shrill screaming and sometimes the wireless. Hears high-pitched sounds.	At age of one year could say "dad", "mum" and "Bob". During the next year made no further progress. Can now say "here" and sometimes "no", but does not say the words he learned earlier.	Hears high tones with right ear but not with left.	
94	4	Until the patient suffered from meningococcal meningitis at the age of 17 months his parents considered his hearing was normal. On the other hand an aunt believes that the child was deaf from birth. He can now hear a band playing, doors slamming, a euphonium, train whistles, wireless and the kick-starter of a motor-bicycle. Imitates a train.	Limited to "mum" and "dad".		
96	2	Partial deafness first suspected by mother at age of 23 months. Apparently hears aeroplanes, wireless, a pianola and train whistles.	At age of 15 months could say "mum", "dad", "Leslie" and "Deidre". Now can say "What's that?" "look", "hullo", "there it is". (Mother has since reported that child, now aged two years 10 months, is speaking fairly well since he has undergone tonsillectomy and syringing of his ears.)	Hears high tones in both right and left ears, but hearing in the latter is slightly better.	Heart disease, concomitant strabismus.
97	2, 11	Deafness first suspected at one year of age. Hears door-bell, telephone ringing, knocking on the door, screaming, hand clapping, a motor-car horn and aeroplanes.	Says "dad", "bub" and "gee-gee". Says "mum" sometimes, but not always appropriately.		
98	4, 8	Hearing apparently good. At two years of age could hear a clock, motor-car horn, motor-car engine, and the telephone ringing. The mother believes that the child hears fairly well, but that he misses occasional syllables in long words.	At one year could say "mumma", "dadda" and "car". He started to speak really well only at three years. Now he can string sentences together and carry on an intelligent conversation. At two years "water" was pronounced "gorki", then it became "gorter" and finally "water".	Child would not cooperate.	
99	4	Deafness first suspected at age of two years. Used to vocalize but would not talk. Hears train whistles, a referee's whistle, hand clapping, a motor-car horn and the telephone ringing.	Says "ta", "please", "mum", "dad" and "Oh, Oh, Oh" in imitation of a train.	No apparent response.	Heart disease.
100	2, 3	Mother first suspected deafness at age of nine months. Can hear water running into a bucket; also shrill noises like "beep-beep". Hears some sounds on wireless, also motor-car horns, but not motor-car engines.	Limited to "mum".	Slight response to high tones in both ears.	
101	3, 7	Although she cannot be perfectly sure, the mother believes that the child's hearing was normal up to the age of 16 months, when he had "gathered" ears. (Apparently, however, only the left ear was affected.) Can now hear the "click" of a light switch, a wristlet watch, motor-car engines, train whistles, squealing and hand clapping. Responds to his name if said loudly. Does not hear people coming up behind him. Hearing is worse when he is out of doors and when he has a cold; hearing improved after adenoidectomy.	Up to the age of 2·5 years made no attempt to speak. Was then taught to say "mum", "Oh" and "Ba-ba" (for "Barbara").	Child would not cooperate.	Heart disease.
102	2	Appears to hear hand clapping and aeroplanes. Takes no notice of the wireless.	Says "dee", "dee", "dee", but nothing else.	Hears high tones and hand clapping in both ears.	Heart disease.
103	3, 6	Hears door banging, motor-cars, a telephone ringing, train whistles, aeroplanes. When out of doors appears to hear up to 12 feet away; at greater distances his response is irregular.	At the age of one year said "mum", "dad", and "ta-ta". Does not often form sentences. Says occasional words such as "hullo" and "baby". Says "Baby is home with Jean". Says "gutton" for "button", "tap" for "hat", "tow" for "cow", "pinger" for "finger", "tenny" for "penny" and "pockie" for "pocket".		Heart disease.
104	2, 5	When the child was aged 10 months the mother noticed that he did not appear to hear the sound of cutlery being dropped. Now hears telephone ringing, the clatter of the front of the wood-stove falling down, the banging of doors and high-pitched sounds.	Limited to "mum-mum".	High tones are heard in both ears, but better in the right.	Heart disease.

TABLE IV.—*Continued.*

Analysis of Congenital Deaf-mutism and Speech-defects following Rubella in Pregnancy.—Continued.

Case Number.	Age at Time of Examination. (Years and Months.)	Hearing.	Speech.	Hearing Tests with Tuning Forks of High (2,048) and Low (512) Pitches.	Associated Defects.
106	4, 3	The child was first thought to be deaf at age of one year, as she did not seem to hear the noise of passing trains. Hears baby crying, her father whistling, her mother calling (on certain notes), aeroplanes, the wireless, and the slamming of a door. Her mother believes the child hears high-pitched sounds better.	Can now say "mum-mum", "bubbs", "pie" and "but" (for "butter")	Hears high tones in both right and left ears.	
109	4, 9	According to her mother, she was not deaf at birth. At the age of one year she suffered from a throat infection and was deaf for three or four days. At the age of 2·5 years she had anorexia, languor and loss of weight. She showed lack of interest and settled partially deaf. Her mother had to shout at her. Since tonsillectomy and adenectomy at the age of three years seven months her hearing has been good, but her mother thinks that hearing is imperfect in the right ear.	Apparently normal.	Deaf in right ear. Hearing good in left ear. Right tympanic membrane looks abnormal, the left normal.	Heart disease.
110	4, 4	Mother believes hearing is good. Apparently hears most normal sounds.	At the age of one year was able to say "mum" and "dad". Now says a few sentences such as "Is that a plane, mum?" "Cannot say "Uncle Len". Sometimes speech is imperfect: thus he says "Lulla" for "Ella", "granyad" for "grandad". For "motor-car" says "bidda-car".	No apparent response.	Heart disease.

instances in the third month, in one instance in the fourth month and in one instance in the fifth month. The average duration of pregnancy in 21 of the cases of deaf-mutism in which the diagnosis was unequivocal was 1·95 months. Clinically the defects of hearing fell into three main classes. The first group, fortunately small, consisted of three children (Cases 78, 84 and 86) who were apparently stone-deaf. In the second group (19 cases) deafness was not total, and the children's residual hearing was sufficient to enable them to detect such sounds as whistling, motor-car and aeroplane engines, certain tones on the wireless, the telephone ringing, motor-car horns and the shrill voices of other children. Speech was often limited to monosyllables such as "mum", but occasionally the child was able to say a few words. Attempts to form sentences, as in Case 103, were rare. With regard to the third group, in Case 80 the character of the speech-defect was compatible with the view that the child had small "islands" of deafness. This may well be also the explanation of the retardation of and defectiveness in speech in Cases 96 and 98, and perhaps also in Case 110. With one exception (Case 109), the external canal and tympanic membrane were normal. On this account, and because of a history suggestive of *otitis media*, doubt must exist in Case 109 as to the part played by rubella in the ætiology of the deafness. (Incidentally in Case 94 there is a history of meningococcal meningitis as well as of maternal rubella.)

The hearing tests adopted (the use of tuning forks of high [2,048] and low [512] pitches) were relatively crude. The results were often vitiated by failure of the patient to cooperate. This lack of cooperation was due partly to the age of the patient, but more often to the strangeness of the surroundings and to the child's natural shyness. Obviously, as has been pointed out by Carruthers,[9] the children should be tested at intervals as they develop. The tests should be carried out by a person to whom the child is accustomed and with whom he feels at ease. The ideal place would be the school which the child attends for speech therapy. Our limited observations merely serve to confirm our earlier impression that high tones are much better appreciated than low tones, and that just as is the case with the eyes, one ear may be much more severely affected by the infection with rubella than its fellow on the opposite side. We believe that in assessing the extent of the deafness in these young children we have gained much more information from the histories given us by the parents than from our own tests. As soon as possible routine audiometric tests should be carried out. Not only will individual audiograms be of value in the prescription of deaf-aids, but they may also serve to determine minor degrees of deafness (as in our third group) too slight to be detected by ordinary methods.

With regard to education, we believe that the ideal plan is that of the nursery day-school (similar to that started in Western Australia[10]) where kindergarten training is combined with speech therapy. Under these conditions it is possible to take advantage of the valuable formative pre-school period between the ages of three and six years, and yet preserve normal home influences. As far as is practicable, and especially so during the pre-school years, boarding-schools should be avoided, as children lose contact with other members of their families, and return at holiday time to find themselves, as it were, "strangers in their own homes". Our aim should be to minimize rather than to accentuate the child's feeling of being "different", and to encourage him to take his normal place in society. We consider that all the patients of the present series are educable. Indeed, in some cases their intelligence appears to be above the average.

Cardiac Abnormalities.

The series consisted of 19 children, of whom 15 were males and the remainder females (Table V). Two of them (Cases 77 and 108) suffered from heart disease without other apparent defect. In addition to the cardiac condition, one child (Case 85) had unilateral cataract and deaf-mutism, 13 (Cases 78, 79, 80, 81, 84, 89, 96, 99, 101, 102, 103, 104 and 109) suffered from either deaf-mutism or deaf-

TABLE V.

Analysis of Cardiac Defects in Babies following Rubella in Pregnancy.

Case Number.	Signs and Symptoms of Heart Disease.	Report on X-ray Examination.	Associated Defects.
75	Loud blowing systolic murmur all over precordium, most marked in the pulmonary and mitral areas.	"Congenital abnormality of the first left rib. No enlargement or deformity of the heart outline. There are some increased markings in the left mid zone which may be vascular, but infiltration cannot be excluded." (A later X-ray examination showed that the increased markings in the mid-zones were vascular.)	Pyloric stenosis.
77	Thrill all over precordium. "Machinery" murmur at all areas, perhaps most marked over pulmonary area.	"The transverse diameter of the heart is increased, indicating general enlargement without any characteristic deformity."	——
78	No clinical evidence of heart disease.	"The transverse diameter of the heart is increased, the appearance suggesting right ventricular enlargement."	Deaf-mutism.
79	Faint, soft, blowing, systolic murmur at the base.	"No abnormality detected."	Deaf-mutism.
80	No clinical evidence of heart disease.	"Slight enlargement of the transverse diameter of the heart, with prominence in the left middle contour, probably a congenital abnormality."	Deaf-mutism.
81	No clinical evidence of heart disease.	"The cardiac outline is enlarged and globular, suggesting a congenital lesion."	Deaf-mutism.
84	No clinical evidence of heart disease.	"The heart outline is globular with filling-in of the waist on the left with congenital deformity."	Deaf-mutism.
85	Thrill present. Loud "machinery" murmur all over precordium, maximal in pulmonary area.	"The cardiac shadow is definitely increased in size."	Unilateral cataract. Deaf-mutism.
89	No clinical evidence of heart disease.	"The cardiac shadow is enlarged in the transverse diameter a little and the hilar shadows are somewhat congested, suggesting the possibility of a congenital lesion."	Deaf-mutism.
96	Soft blowing systolic murmur at base, most marked in pulmonary area. Can be heard better when the child extends his head.	"No abnormality detected."	Deaf-mutism, strabismus.
99	Faint, soft, blowing, systolic murmur over the mitral area.	"The cardiac shadow is a little enlarged in the transverse diameter with the waist somewhat filled in."	Deaf-mutism.
101	Thrill present. To-and-fro murmur detectable all over precordium. The murmur has a definite "machinery" character, especially in the pulmonary area. Possibly patent *ductus arteriosus*.	"The cardiac shadow is enlarged, suggesting a congenital heart lesion, but there are no radiological signs to indicate the nature."	Deaf-mutism.
102	Faint systolic murmur in mitral area, especially after exercise. The murmur was not detectable on re-examination five weeks later.	"The heart has a mitral configuration with slight increase in the transverse diameter."	Deaf-mutism.
103	No clinical evidence of heart disease.	"The cardiac shadow is slightly enlarged."	Deaf-mutism.
104	Slight roughening of the second sound in the pulmonary area.	"There is no enlargement of the heart outline, though there is some filling in of the waist of the heart."	Deaf-mutism.
108	No clinical evidence of heart disease.	"The heart is definitely enlarged. No aortic knob is visible, the appearance suggesting a congenital abnormality."	——
109	No clinical evidence of heart disease.	"No enlargement or deformity of cardiac outline although the aorta is not visible and the waist filled in slightly."	Deafness.
110	Faint systolic murmur at the apex in certain phases of respiration.	"The pulmonary arteries are prominent, but the heart is not enlarged or otherwise deformed."	Speech defect.
113	No clinical evidence of heart disease.	"The heart is not enlarged, but there is deformity of the right outline suggesting a congenital defect."	Mongolism.

ness, one (Case 110) had a speech defect, one (Case 75) had pyloric stenosis and one (Case 113) was a mongol.

None of the patients was cyanotic. Questioning of the mothers disclosed that in only four instances (Cases 75, 78, 80 and 84) had cyanosis been observed. Usually it was noted soon after birth and was slight and fleeting. In this connexion it may be stated that cyanosis of similar character occurred also in Case 92, in which there was no evidence of heart disease.

A thrill was detected in three cases (Cases 77, 85 and 101). In each instance it was associated with a systolic-diastolic murmur, which had a "machinery" character and was most pronounced in the pulmonary area. There was also radiological evidence of abnormality, but this was not sufficient to assist in the diagnosis of the exact nature of the defect. Most probably, however, these cases are examples of patent *ductus arteriosus*.

Bruits were also heard in six other children; in Cases 75, 79, 96, 99 and 110 the murmur was systolic and in Case 104 it was diastolic. In three of these cases (Cases 75, 79 and 96) the results of X-ray examination were negative, and in the remainder (Cases 99, 104 and 110) suspicious or doubtful.

In the remaining cases of the series (Cases 78, 80, 81, 84, 89, 102, 103, 108, 109 and 113) although clinical examination gave negative results, the X-ray findings indicated or suggested a congenital abnormality. We,[10] and later, New South Wales workers,[12] had previously noted a similar discrepancy. We agree with these investigators[12] that the reports should be accepted with a degree of caution. On the other hand, such observations may indicate that, just as is the case in the diagnosis of pulmonary tuberculosis, auscultation and other forms of physical examination may sometimes be inferior to radiography. (We wish to emphasize that with the object of eliminating artefacts all the X-ray examinations were carried out at a distance of six feet.)

In our opinion, in at least five cases of the present series (Cases 79, 102, 104, 109 and 110) the diagnosis should be looked upon as doubtful. Further examination at intervals as the children grow older may perhaps eliminate these and other cases.

Microcephaly.

In seven instances (Cases 77, 78, 80, 86, 88, 89 and 97) the head circumference measured between 1·0 and 1·8 inches less than normal, and in 13 instances (Cases 79, 82, 84, 85,

87, 92, 95, 100, 101, 102, 105, 106 and 113) it measured between 0·4 and 0·9 inch less than normal. Variations from normal of 0·3 inch or less, which were present in a further eight cases, were disregarded. Measurements of similar magnitude were also disregarded in the patients whose head circumferences were larger than normal. Two children (Cases 104 and 109) had head circumferences of 0·4 inch greater than normal, while in the remaining three instances (Cases 75, 93 and 96) the children's respective head circumferences were 0·5 inch, 1·6 inches and 2·5 inches greater than the average. In Case 95 microcephaly was the only abnormality detected, while in Case 105 this defect was accompanied by backwardness in development.

Other Abnormalities.

There were two cases (Cases 75 and 114) of hypertrophic pyloric stenosis; in the former the defect was associated with heart disease, in the latter other anomalies included spastic diplegia, right inguinal hernia and strabismus. The child in Case 76 had a cleft palate. *Spina bifida occulta* was present in Case 112. In addition to deaf-mutism, the patient in Case 87 suffered from an occipital nævus.

Absence of Congenital Defects following Rubella just Prior to Pregnancy.

Two mothers (Cases 115 and 116) suffered from German measles about thirteen days and six days respectively before conception. (At the present time it is generally accepted that ovulation occurs approximately fourteen days before the onset of the next menstrual cycle, and that fertilization takes place within twenty-four to forty-eight hours of ovulation.)

In Case 115, in view of the possibility that the child might be malformed, therapeutic abortion was carried out when the mother was about three and a half months pregnant. The specimen sent to us consisted of fragments of tissue. Both eyes, however, were intact, and we were also able to identify the right inner ear (which had been "shelled out" of the temporal bone), a kidney and suprarenal gland, and fragments of the heart, the liver, the spleen and the intestines. Histopathological examination of these specimens revealed no abnormality.

In Case 116, on physical and X-ray examination the child was normal.

Morbilli During Pregnancy and Congenital Abnormalities.

The series consisted of eight cases (Cases 117 to 124 inclusive) (Table I). One mother (Case 117) had contracted morbilli in the first month of pregnancy, one (Case 118) in the second month, two (Cases 119 and 120) in the third month, three (Cases 121, 122 and 123) in the fourth month, and one (Case 124) in the eighth month. With two exceptions (Cases 121 and 123) the infants born subsequently were normal. In Case 121 the child suffered from pyloric stenosis. Clinically the heart was normal. The X-ray report, however, was as follows: "Cardiac shadow is unusual and broad in the region of the great vessels. This is suggestive of a congenital lesion and there may be in addition persistent thymus." In Case 123 the child suffered from *genu valgum*.

Mumps During Pregnancy and Congenital Malformations.

Three mothers had contracted mumps during pregnancy, two of them (Cases 125 and 126) during the first month and one (Case 127) during the seventh month. The only abnormality in the child born subsequently in Case 125 was a slight capillary nævus of the right upper eyelid. In Case 126 there was a hint of an anal dimple, but the true anus was absent and the large intestine terminated in a passage which opened inside the *labia majora*. The head circumference in Case 127 was slightly below normal.

Varicella During Pregnancy and Congenital Anomalies.

There were two cases (Cases 128 and 129) in which the mother had suffered from chicken-pox in pregnancy, in both instances in the seventh month. One of the infants born subsequently was normal, the other (Case 128) had a nævus of the scalp and radiological signs suggestive of a cardiac defect. The report read: "The heart is not enlarged, the aorta is small and the configuration suggests right ventricular preponderance."

Herpes Zoster During Pregnancy and Congenital Defects.

Two mothers had had shingles in pregnancy, one of them (Case 130) during the fourth month, and the other (Case 131) during the sixth month. In the latter case the child born subsequently suffered from a myelocele, bilateral talipes and right *genu recurvatum*.

The congenital defects in Case 130 included deaf-mutism, microcephaly and heart disease. The diagnosis of the cardiac defect was based on the presence of a harsh systolic murmur all over the precordium, but most apparent over the pulmonary and mitral areas; the second sound was "slapping" in type. The X-ray report was as follows: "The heart shadow is considerably enlarged with upward displacement of the pulmonary artery, filling in of the waist and increased vascular markings in the lungs." The pronounced resemblance of the abnormalities in this case to those associated with rubella in pregnancy prompted us to question the mother more closely. We found that when she was two months pregnant the husband had suffered from German measles (diagnosed by a medical practitioner). Shortly afterwards she suffered from sore eyes, but did not develop a rash. In this connexion it may be mentioned that Dukes[37] considered that suffusion of the conjunctiva—"pink eye"—might be the only symptom of an attack of rubella. It is feasibile, therefore, that in this case the ætiological factor may have been sub-clinical rubella.

Scarlet Fever During Pregnancy and Congenital Abnormalities.

One woman (Case 132) developed scarlet fever when seven months pregnant. The child born subsequently was below average birth-weight, and his skin was wrinkled, so that he had an "old man" appearance. The jaundice, present at birth, increased in intensity and was associated with the passage of pale fæces. Death occurred at the age of five weeks. Post-mortem examination disclosed congenital obliteration of the bile ducts.

Discussion.

Doubt has been expressed whether or not the exanthematous disease contracted by the mother in pregnancy[1,2,3,4] is true German measles. Careful scrutiny of the symptomatology in the present series of cases fully corroborated our earlier conclusions. Admittedly, in the intensity of the prodromal and concomitant general constitutional symptoms, and also in the development of sequelæ such as arthritis, the clinical picture differed somewhat from that considered to be typical of rubella.[38,39] Evidence[39] is accumulating, however, that German measles may be altering in character to a more severe form.

Some critics have suggested that the association between rubella in pregnancy and congenital defects may be purely fortuitous. Against this view there are a number of strong objections. The first is the large number of cases—over 400—in which the relationship has been recorded. Even more convincing is the fact that in these cases the period at which the mother suffered from German measles is virtually confined to the first four months of pregnancy, whereas if the association was fortuitous it would be reasonable to expect a more even distribution over each of the nine months. Finally, if the relationship was purely coincidental, a considerable range of abnormalities might be anticipated. Analysis of the defects in the present and earlier cases[1,2,3,4,5,6,7,8] shows, however, that the range is extremely limited. With few exceptions, the malformations are reproductions of varying degrees of completeness of a specific syndrome, comprising cataract, deaf-mutism, heart disease and microcephaly. The complete syndrome is uncommon, as cataract and deaf-mutism are rarely encountered together. The commonest types of defect consist of deaf mutism or cataract, either alone or in combination with one or both of the remaining components of the syndrome.

With regard to the mechanism of production of congenital anomalies, Stockard,[40] on the basis of extensive experi-

ments with the eggs of the common minnow *Fundulus heteroclitus*, came to certain conclusions, which Mall[20] believed were equally applicable to the genesis of congenital malformations in human beings. Accordingly it seemed of considerable interest to ascertain how far the results of the present and earlier work on rubella and congenital defects supported Stockard's views. We shall consider each of Stockard's conclusions in turn. The first is as follows:

> The type of abnormality is determined by the particular developmental "moment" at which the noxa acts. At different periods in the development of the ovum, certain primordia are undergoing rapid proliferation, and may be thought of as developing at a rate entirely in excess of the general developmental rate of the embryo. At such periods these primordia are peculiarly susceptible to unfavourable influences, while only slight or no ill effects may be suffered by the embryo as a whole.

We found[9] that in our cases of deaf-mutism the average duration of pregnancy at the time of the maternal infection with rubella was 2·3 months, whereas in the cases of cataract it was 1·5 months. This suggested to us that the type of congenital defect was dependent upon the stage of pregnancy at which the mother developed German measles. (With greater experience we have realized that except when the date of commencement of the last menstrual period and the date of appearance of the rash are known, estimation of the precise period of gestation at which the mother suffered from rubella is subject to certain inaccuracies. In our opinion the majority of mothers calculate their stage of pregnancy from the first day of the last menstrual period, others from the last day, and a few from the seventh day after the onset of the last menses. Assuming that each mother was approximately ten days (0·3 month) out in her reckoning, we consider that the above figures should be modified to 2·0 months in the case of deaf-mutism and to 1·2 months in the case of cataract.) Mann[11] considered that there was a definite parallelism between our statistics and the known times of active proliferation of the lenticular and cochlear *Anlagen*. In Case 91 of the present series, in one eye was found lack of closure of the fœtal (chorioidal) fissure, which normally closes during the seventh week.[12] In this instance the mother had developed rubella during the seventh or eighth week of gestation. Thus here, too, the defect and the inferred time of attack of the virus were complementary. Carruthers,[9] while concurring that the stage of pregnancy at which the mother contracts rubella is of significance in determining the nature of the developmental anomalies, considers that the taking of an average as we have done may be misleading. As he points out, deaf-mutism, for instance, may result from maternal rubella in several of the earlier months of pregnancy. The obvious conclusion, however, is that in man the primordia of various organs have relatively long so-called "critical moments". Our experience suggests that in general the "critical moment" of the optic primordium is the first three months of pregnancy and that of the cochlear *Anlage* is the second, third and fourth months. We believe, moreover, that averages such as we have drawn above may indicate the period of greatest susceptibility of the *Anlage* of a particular organ—for example, lens or cochlea—to the virus of rubella.

It is evident, however, that there must be other factors at work in determining whether or not a particular primordium is affected, even if it is rapidly proliferating and has developmental "precedence" over other centres of growth. For instance, it is hard to understand why unilateral cataract should occur, because the *Anlagen* of the lenses must surely have reached the same stage of growth, and therefore might reasonably be expected to be equally susceptible or immune to the virus. Similarly, in Case 91, why should the fœtal fissure be closed in one eye and open in the other?

Finally, we wish to refer briefly to another point in Stockard's first conclusion—namely, that while at the "critical moments" certain primordia are particularly sensitive to various types of injury, only slight or no ill-effects are suffered by the embryo in general. The fact that many of the children suffering from congenital defects as the result of maternal rubella also exhibit stunting of growth, indicates that as well as the main attack of the virus localized to the specialized cells of certain primordia, there is a general but much slighter attack upon the less-specialized cells of the skeletal tissues.

Stockard's second conclusion is as follows:

> The earlier the arrest the more numerous will be the types of defect found, and the later the arrest the more limited the variety of deformities, since there are fewer organs to be affected during their rapidly proliferating primary stages.

On this point the evidence gathered so far is equivocal. In our own cases, for instance, whether the mother contracted the disease in the first month or the second month of pregnancy seemed to be of little significance in determining the number of congenital defects in the children born subsequently. We consider it worthy of note, however, that when the New South Wales committee[12] found the abnormalities, deaf-mutism, eye disease and heart disease in combination in the one child, in all instances the mothers had contracted rubella within the first two months of pregnancy. Moreover, six of the eight mothers had contracted the disease during the first month of pregnancy. On the other hand, attention should be drawn to the fact that the committee and ourselves have both observed children with single congenital defects resulting from maternal rubella in the first month of gestation.

Stockard's conception gains support from the opinion of Carruthers.[9] This author makes the following statement:

> I am inclined to think that the importance of the stage of pregnancy at which infection occurs is that, if it is in the first six weeks, fœtal damage will be widespread and may include the eyes, both divisions of the ears, the heart and perhaps many other parts. After the sixth week the eyes may escape, the heart may be spared and the semicircular canals may become normally developed; but the cochlea is still likely to be damaged, and growth may be retarded. After the third month damage to the fœtus is rare.

This view should be modified, because our figures and those of the New South Wales committee[12] show (i) that deaf-mutism subsequent to maternal rubella in the first month of pregnancy is relatively uncommon, and (ii) that the contraction of rubella in the fourth month of pregnancy is relatively often followed by congenital malformations.

Stockard's third conclusion is the following:

> If development is arrested or retarded at a stage when no unusually rapid changes are taking place, a comparatively quiescent moment during which all parts are developing, but during which no particular or important part is proliferating at an excessively high rate, the embryo may escape injury.

We believe that this conclusion of Stockard is the logical explanation of the non-occurrence of congenital abnormalities in babies whose mothers have been infected with German measles after the fourth month of pregnancy.

If such "moments of indifference", as Stockard calls them, occurred in the earlier months of pregnancy, they would serve to account for the (so far) rare cases in which mothers have contracted rubella during these months and yet have given birth to apparently normal infants. Whether this interpretation is correct remains unproven. It seems to us just as likely that in such instances the virus fails to penetrate the barriers between mother and embryo and therefore has no chance of attacking the latter.

Our earlier figures[9,13] suggested that if a woman suffered from rubella in the first two months of gestation, the chances of her bearing a congenitally malformed child were in the region of 100%. In the present series we have reported one case (Case 93) in which the mother contracted the disease at the end of the second month, and yet gave birth to an apparently normal child. A similar case has been described by the New South Wales committee,[12] in which rubella occurred during the first month of pregnancy. (In this instance the disease was not diagnosed by a medical practitioner.) We agree with these observers that such findings ". . . may indicate that an attack of rubella

during the very early months of pregnancy is not invariably followed by the presence of congenital defects in the child".

The following is Stockard's fourth conclusion:

> The same abnormality may be induced in embryos by a great number of different noxa.

Ida Mann[11] states that ". . . it may well be found that a variety of other causes operating during the first six weeks (of pregnancy) may produce congenital cataract and heart disease. If this result is found to be absolutely specific for rubella it will be surprising from an embryologist's point of view". However, as she points out ". . . the placenta acts as a partial barrier to the passage of certain noxious substances to the embryo. . . . Therefore, before assessing the effect on growth of a toxin of any sort in the maternal blood in man we must know whether the placenta is pervious to it or not."

Previously, in conjunction with our colleagues[12] we reported seven cases of morbilli, most of them occurring relatively late in pregnancy, one of which was followed by spontaneous abortion, while the remainder were followed by the birth of normal infants. In the present series we have recorded a further eight cases; two of these mothers later gave birth to babies with malformations. We consider it doubtful whether morbilli played any rôle in the ætiology. It is noteworthy that in the four instances in which the infection was contracted in the first three months of pregnancy (the period of greatest susceptibility of the embryo), the babies born subsequently were normal. With regard to the congenital anomalies we have recorded in association with the occurrence of maternal mumps, varicella, *herpes zoster* and scarlet fever, with the exception of the cases of mumps, in most instances the infection occurred late in pregnancy; this suggests that the relationship was merely fortuitous.

Obviously, records of a considerable number of cases will need to be compiled before it can be ascertained whether or not infectious diseases other than German measles in pregnancy are responsible for the development of congenital abnormalities. We agree with Ida Mann that an important factor in determining such a relationship may prove to be the penetrability or otherwise of the barrier between mother and embryo. As far as smallpox, chickenpox and morbilli are concerned, however, there is evidence that they are capable of penetrating the placental barrier late in pregnancy. Whether the same condition obtains early in pregnancy is as yet unknown.

Nevertheless we concur with Ida Mann that there is a considerable degree of parallelism between the results of experimental embryology and the available data on congenital malformations following maternal rubella in pregnancy. Such divergences as may later become evident will, we think, be dependent on the fact that investigators such as Stockard were concerned with inanimate noxa (reduction of oxygen supply, lowering of temperature, treatment with alcohol or with ether), whereas we are dealing with a living virus able both to proliferate and to attack living cells. In this connexion we may mention that we have been puzzled by the occurrence of deaf-mutism and cataract in combination in the same child as the result of maternal rubella in the first month of pregnancy.[13] Our figures and those of the New South Wales committee[13] show that during this period, while the optic *Anlage* is susceptible to the virus, the cochlear *Anlage* is virtually insusceptible. If, however, it is assumed that the virus of rubella may persist in the tissues of the embryo for some time after its initial invasion, then it would be possible for it to attack each primordium (eye, ear, heart *et cetera*) as it reached its so-called "critical moment". As one of us has already noted,[12] the fact that secondary as well as primary lens fibres are involved in the cataract suggests a continuation of the pathological process for some time after its onset—that is, a persistence of the virus of rubella in the tissues. Finally, we should like to point out that Stockard considered that all congenital anomalies were the result of arrested development; he was entirely unable to agree with Mall that the arrests must usually be associated with actual destruction of tissue. Stockard's views may well be correct for inanimate noxa, but as far as rubella is concerned our limited pathological findings[12] tend to support Mall.

Mongolism was observed once in the present series (Case 113). The mother had suffered from German measles when she was seven or eight months pregnant. As one of us[12] has previously pointed out, the association was probably fortuitous, as experience shows that the occurrence of rubella late in pregnancy is unlikely to result in abnormalities. Incidentally the fact that mongolism is often complicated by congenital heart disease and by cataract suggests that whatever the causative agent of mongolism may be, it is operative in the early months of pregnancy.

Earlier[12] we mentioned that Gregg recorded a case of congenital cataract in which rubella was said to have been contracted by the mother three months before the onset of pregnancy. We also described a doubtful case. In the present paper we have reported two instances (Cases 115 and 116) in which rubella just prior to conception failed to lead to congenital abnormalities. In our opinion, there is as yet no evidence to justify therapeutic abortion in cases in which the disease is contracted a short time before fertilization. On the other hand, we consider that in the absence of effective prophylaxis, therapeutic abortion is entirely justifiable in the case of any woman who suffers from German measles in the first four months of pregnancy.

Summary.

In the course of the present investigation 56 infants and two fœtuses were examined; 46 of them were found to have congenital malformations.

In 40 instances the mothers had suffered from rubella in pregnancy; 36 of the infants and a fœtus exhibited congenital defects. The abnormalities comprised 11 cases of deaf-mutism, 11 cases of deaf-mutism and heart disease, one case of deafness and heart disease, one case of deaf-mutism, heart disease and strabismus, one case of deaf-mutism, cataract and heart disease, one case of deaf-mutism and nævus, one case of speech defect and heart disease, one case of cataract and lack of closure of the fœtal fissure, two cases of heart disease, one case of mongolism and heart disease, one case of microcephaly, one case of microcephaly and backwardness in development, one case of cleft palate, one case of *spina bifida occulta*, one case of heart disease and hypertrophic pyloric stenosis, and one case of spastic diplegia, hypertrophic pyloric stenosis, inguinal hernia and strabismus. In addition, in a further 18 of the foregoing cases microcephaly was also present. Four of the mothers had had German measles in the first month of pregnancy, 19 in the second month, eight in the third month, two in the fourth month and one in each of the fifth, sixth and eighth months; in the remaining case the duration of pregnancy at the time of infection was not determined. In three cases in which the infant born subsequently was normal, the mothers had contracted the disease in the second, fourth and sixth months of pregnancy, respectively.

In two instances in which rubella was contracted less than a fortnight before conception, the offspring were apparently normal.

The infectious diseases during pregnancy in the remaining 16 cases included eight cases of morbilli (two of the babies were abnormal), three cases of mumps (all babies had defects), two cases of varicella (one baby was malformed), two cases of *herpes zoster* (both babies exhibited abnormalities), and one case of scarlet fever (the baby was defective).

The pathogenesis of the anomalies is discussed in the light of the experiments of Stockard.[11]

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CŒLIAC DISEASE: A SURVEY FROM THE CHILDREN'S HOSPITAL, MELBOURNE.[1]

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CŒLIAC DISEASE was first described by Samuel Gee[(1)] in 1888. Since then it has been variously known as "Gee's disease", "acholia", "intestinal infantilism", "Gee-Herter disease", "Herter-Heubner disease" and "Gee-Thaysen disease". Cœliac disease is a chronic disorder of early childhood, characterized by the passage of large, pale, fatty and offensive stools, and accompanied by severe wasting and—at the time of the illness at least—much interference with bodily growth. It is not commonly met with in childhood, but is found in all grades of society. It is generally said to affect girls a little more frequently than boys. This paper is a review of thirty-five cases of cœliac disease at the Children's Hospital, Melbourne, during the years from 1933 to 1943, inclusive, together with some account of the modern outlook on this affection.

Historical.

The word "cœliac" is difficult to trace with certainty. It probably derives from the Latin word "*cœlum*", which is connected with a Greek word meaning "hollow". The meaning in Latin is "the heavens", and on the authority of "Cassell's Latin Dictionary", Cicero used the word in connexion with climate, to mean "unhealthiness". As Gee knew that the signs of the disorder he described were at times to be found in those who had lived in India, there may have been some connexion in his mind between climate and what he designated the "cœliac affection". In any event, the term "cœliac disease" is probably a misnomer. There are two reasons for this assumption. In the first place, careful study has not, by any methods of investigation at present available, revealed any disease process in the bowel, which is apparently affected in some way. However, the future may reveal some discovery whereby the tissues may be found to show some changes of significance. In the second place, the symptoms and signs of cœliac disease may follow more than one cause, so that we should more correctly speak of the "cœliac syndrome". For example, wasting accompanied by pale, frothy stools may result from such diverse affections as pancreatic fibrosis, whereby there is a deficiency of digestive ferments, or from mesenteric glandular tuberculosis, involving obstruction to free flow along the lacteals passing from the bowel wall. Such a conception as a syndrome to include this disorder and others is not a novelty. As a similar instance, the case of idiopathic pernicious anæmia may be mentioned. This disease is now recognized as having a blood picture common to more than one disorder, and the causes exhibit such diversity as a deficiency in diet and surgical resection of the bowel. As in pernicious anæmia, so in cœliac disease, investigation has revealed a number of causative factors to explain the same or very similar findings.

The study of cœliac disease may be said to have commenced with a paper on the "cœliac affection", written by Samuel Gee.[(2)] Gee described what he termed "a kind of chronic indigestion which is met with in persons of all ages, yet is especially apt to affect children between one

[1] Read at a meeting of the Melbourne Pædiatric Society on August 8, 1945.

and five years old". Gee gave a vivid and accurate description of the stools, which had the appearance of oatmeal porridge or gruel. Gee noticed that wasting was more evident in the limbs than in the face. He observed that emaciation was at first more apparent to the hand than to the eye, and that the flesh had a soft, flabby feel. He remarked on the great muscular weakness and tenderness often present. Gee was aware, as has formerly been mentioned, that certain subjects exhibiting cœliac disease, whose condition he described, had lived in India at one time. Many years later Thaysen[2] drew a destinction between what was called tropical sprue and idiopathic steatorrhœa, a name which he gave to fatty diarrhœa in all cases in which there had been no residence in the tropics. To Thaysen, non-tropical sprue in adults and cœliac disease in children were essentially identical conditions. This view has been widely accepted, notably by Bennett, Hunter and Vaughan.[4]

Bramwell,[5] in 1902, described a case of adolescent infantilism, with fatty stools; he claimed that he had effected improvement by the use of pancreatic extracts, and Cheadle[6] in the following year reported five cases of "acholia" in children and one case of tropical sprue. Cheadle attributed the condition to "inhibition of the liver". Later, Bramwell noted swelling of the abdomen and tympanites. He observed also non-union of epiphyses, anæmia, and tetany and achlorhydria. He postulated "pancreatic infantilism" as the cause, but his conclusions were not supported by post-mortem evidence.

In 1908, Herter[7] published "Infantilism and Chronic Intestinal Infection". He observed the arrested bodily development, with good mentality, and the abdominal enlargement. He noted physical and mental fatigue, with anæmia, and the gastro-intestinal upset. Herter stressed intestinal infection as the cause of the symptoms and signs observed. Indeed, Herter anticipated much of what followed in biochemical investigations, by bringing to notice the high fæcal fat content and the loss of fatty acids in the stools, by his studies of the calcium balance, and by noting the carbohydrate intolerance as opposed to the better utilization of protein in cœliac disease. However, Herter's views in regard to infection have not been supported by later work. Heubner,[8] inspired by Herter's work, also published his studies in 1909.

The beginning of a changed outlook on cœliac disease occurred in 1926, when Thaysen,[9] in cases of non-tropical sprue, observed an abnormally small rise in the blood sugar level after ingestion of glucose. Thaysen defined a low blood sugar curve as one showing a rise of 40 milligrammes per 100 millilitres of blood or less during the two hours following the ingestion of 60 grammes of glucose by adults. Parsons[10] later pointed out that it was more important to observe the absence of or great delay in the occurrence of a true peak in the curve, and the failure of the curve to fall to the fasting level in two and a half hours, even after a small rise. Thaysen concluded that the low blood sugar curves observed in cœliac disease were not due to defective absorption or to destruction of dextrose in the intestinal tract, but to a toxic effect on the endocrine glands controlling the blood sugar level in the blood. Thaysen administered glucose by the intravenous route, as well as by the mouth, and observed that the resulting blood sugar curves were as often low after the former as after the latter method had been used. He also undertook observations on the respiratory quotient, and found a rise to about one after ingestion of glucose. Furthermore, the respiratory quotient rose higher when a carbohydrate diet was taken than when an ordinary diet was taken. In regard to Thaysen's first observation—namely, that the rise in blood sugar level is small as often after intravenous administration of glucose as it is after ingestion of glucose by mouth, Parsons[10] offers a possible explanation. He considers that the rate of absorption of glucose depends in the first place on the rate at which sugar enters the blood, and in the second place on the rate of removal of glucose from the blood, or the glucose tolerance of the subject. The first may be affected by gastric stasis or by failure of absorption. Thus impaired absorption will lead to the presence of less sugar in the blood, and a low blood sugar curve will result. The carbohydrate deprivation thus produced results in impaired tolerance, according to the degree and duration of the absorptive disability. If this disability is passed, then the curve will be higher, and again lower as tolerance improves. Thus the curve always represents a resultant of two factors—namely, absorption and tolerance. Contrary to Thaysen's conclusions, subsequent investigation followed the theory that some absorptive disability was responsible for the symptoms and signs of cœliac disease.

Modern Trends.

Ross,[11] in 1936, observed the blood sugar curves after administration of glucose by the oral and intravenous routes. He demonstrated a flat glucose curve after oral administration with a high curve after intravenous administration in cœliac disease, chronic intestinal indigestion and abdominal glandular tuberculosis in children, and concluded that this resulted from deficient power to absorb carbohydrate from the intestine. Ross postulated that tolerance of a subject for glucose was directly proportional to sensitivity to injected insulin. The relation being linear, glucose tolerance was sensitive to endogenous insulin. Again, insulin sensitivity was due to insulin "kinase". Finally, glucose tolerance was found to depend on the amount of carbohydrate contained in the diet during the period preceding the test. This followed from the previous postulates, the supposed "kinase" being formed in the liver. Crude liver preparations, such as "Campolon", were found to improve glucose tolerance, whereas more refined preparations, such as "Anahæmin", proved to be less effective. Later, Himsworth,[13] who originated the idea of "kinase", came to doubt its existence, and postulated an endocrine factor in the nature of an anti-hormone.

The problem was further studied by Crawford[12] in 1938. Crawford considered the following causes of a low blood sugar curve in cœliac disease: (i) a lowered renal threshold for glucose; (ii) a diminished or delayed absorption of sugar from the bowel; (iii) an increased rate of utilization or storage of glucose in the tissues after absorption.

Crawford carried out intravenous glucose tolerance tests in 12 cases of cœliac disease, in 11 of which the blood sugar curves after oral ingestion of glucose were typically low. No significant differences were found from normal subjects. In five there was no difference between the results in the active and in the latent phases of the disease. Further, insulin sensitivity tests on six of the patients gave results closely similar to those obtained from six normal test subjects. Crawford concluded that there was no abnormality of intermediate carbohydrate metabolism in cœliac disease, and therefore, by exclusion, that the low curves were due to delayed or defective absorption of carbohydrate from the bowel. This finding presumably applies also to other forms of idiopathic steatorrhœa, and to cœliac disease as well.

In 1940, May and McCreary,[14] of Boston, studied the glucose tolerance test in association with radiological studies relating to intestinal motility. Low blood sugar curves were found in association with "clumping" of the barium bolus in views of the small intestine. The absorptive defect was regarded as dependent on defective motility of the bowel, in the absence of any specific defect of the intestinal mucosa in cœliac disease. "Clumping" and low blood sugar curves are not, however, diagnostic of cœliac disease, being found also in disease of the pancreas, sprue, cretinism and infections, and occasionally in normal subjects.

The factor described by Bennett and Hardwicke[15] in 1940 was the next stage in the elucidation of the cause of cœliac disease. This was to postulate a chronic jejuno-ileal insufficiency, which embraced many substances other than carbohydrate. Thus, given some factor, as maintained by Bennett and Hardwicke, the meteorism and frothy stools, for instance, of cœliac disease, could be explained by defective absorption of the *succus entericus*. A long list is provided by these writers; it includes the three basic foodstuffs—namely, protein, carbohydrate and fat (deficient absorption of fat, for instance, producing wasting

and steatorrhœa). Iron and the anti-pernicious-anæmia factor are not forgotten; and so to the vitamins. In the vitamin-deficiency group, the most prominent condition will obviously be rickets. Even vitamin *E* is mentioned, when it is asserted that sterility and infantilism may result should this vitamin fail to be efficiently absorbed from the intestine. Perhaps this classification of absorptive defects will serve to draw attention to the wide clinical field embraced in the syndrome of cœliac disease; but it is too artificial to be seriously considered in relation to a clinical study.

More important is the study of May, McCreary and Blackfan,[38] who follow on from their work with radiology referred to above and tested out the absorption of glucose from the small intestine in association with the absorption of fat and of vitamin *A* in normal subjects. These results were then compared with the findings in cœliac disease, and in cases of pancreatic fibrosis. The method of duodenal intubation for the introduction of glucose was used in asociation with a means of stimulating the bowel with mecholyl. May, Creary and Blackfan concluded that the defictive absorption of fat and of carbohydrate in cœliac disease lay in a defect of the intestinal mucosa. Fat and glucose absorption and intestinal motility were improved by the use of crude liver extracts, and of vitamin *B* complex, administered parenterally. Definite clinical improvement resulted. In 1942, Flax, Barnes and Reichert[37] also showed an apparent close relationship between vitamin *A* absorption and gastro-intestinal motility and tone in fibrocystic disease of the pancreas. Furthermore, the biochemical process involved in fat splitting and absorption is under review. It is possible that this work may throw some light on the mechanism of one aspect of the cœliac syndrome.

Probably we must go back to 1938 for the commencement of the important advances in the study of cœliac disease, for it was then that Anderson,[36] as the result of work on pancreatic fibrosis in infants, suggested the classification of cœliac disease and allied conditions as a syndrome, called the cœliac syndrome. This widened the viewpoint at once, and left the way open for real advances, not only in the study of cœliac disease, but in the dietetic problems of infancy and their allied "dyspepsias", which frequently test the patience of the most experienced clinicians.

Anderson analysed 49 cases of pancreatic fibrosis among infants and older children in which a necropsy had been performed. Forty-five of the 49 cases were of cystic disease (fibrosis) of the pancreas. The remaining four were cases in which the acinar tissue of the pancreas was replaced by adipose tissue. Anderson divided her material into three groups as follows:

Group I: Death in the first week of life from some form of intestinal obstruction—five cases.

Group II: Death between one week and six months of age from purulent bronchitis, bronchiectasis or bronchopneumonia. Thirteen of these 19 cases cases were essentially of feeding problems; the subjects failed to gain in weight on an adequate diet, vomiting and diarrhœa were absent, but stools were foul and bulky.

Group III: Death from pulmonary infections between the ages of six months and fourteen and a half years—25 cases; in all the diagnosis of cœliac disease was made.

Anderson found certain definite pathological changes, involving the acinar tissue of the pancreas, the lungs and the small intestine, and evidence of vitamin *A* deficiency in many children who died before the age of one year. The acinar tissue of the pancreas was replaced by epithelium-lined cysts, containing concretions and surrounded by fibrous tissue. The islet areas were intact. In the lungs the usual infecting agent was the *Staphylococcus aureus*, and lung abscess was found, in addition to the conditions referred to above in Group II. All lesions were probably, if not always, present at birth. Sixty-seven *per centum* of the subjects were females, and in three of these cases the disease was of familial occurrence. Anderson further estimated that in 90% of pancreatic tissue in 3·3% of 605 unselected patients admitted to the Babies' Hospital, New York, involvement of the pancreas in the condition of cystic fibrosis was present. Anderson then classified her material in the following way: *A*, patients with pancreatic insufficiency who survive the first year of life; *B*, patients with chronic recurrent diarrhœa who have no essential abnormalities of the pancreas. The final conclusion put forward by Anderson at that time was that cœliac disease was a clinical syndrome, and that differentiation between the above-mentioned two classes could be made with certainty only by examination of the duodenal contents for enzymes, or by microscopic examination of the pancreas. The syndrome could be produced by two or more disease entities, one of which was cystic fibrosis of the pancreas.

In 1942, Anderson[39] published further work and gave the results she had obtained on analysis of duodenal secretions. It is held that assay of trypsin in the duodenal juices is a reliable means of diagnosis in cystic disease of the pancreas, or in the determination of pancreatic deficiency, on account of the fact that trypsin is present in low concentration or may be entirely absent. Congenital pancreatic deficiency, it is held, is therefore readily to be differentiated from other varieties of the cœliac syndrome, in which trypsin is found to be present in the normal concentration. Examination revealed the presence of trypsin in the normal infant's duodenal contents, and also in smaller but appreciable quantities in cases of extreme marasmus. Amylase was found to be present in low concentration during the first few months of life and in many older infants and children suffering from chronic diarrhœa. Amylase was occasionally present in cases of pancreatic fibrosis, and lipase was present in normal infants of all ages. The concentration of lipase, however, was reduced in congenital pancreatic deficiency; but its estimation was less reliable. Following this work, Anderson suggested the following two further groups to be separated off from the general group of cœliac disease: (i) "near" cœliac disease, associated with intolerance of starch and a low amylase content in the duodenum; starch is present in the stools, and there is no excess of stool fat; the clinical course is mild but prolonged; (ii) a class of idiopathic steatorrhœa, associated with intolerance of fat, high concentration of stool fat, normal pancreatic enzymes, and a severe and protracted clinical course. Anderson cites three cases of severe marasmus, in which the trypsin, amylase and lipase levels in the duodenal contents were depressed, but returned to normal levels with the onset of clinical improvement. The depression of pancreatic function was supposed to be due to the lowered general state of the organism; but it may form part of the clinical picture owing to the consequent difficulty in digestion. This was suggested also by the bulk of and the excess of fat in the stools of two of the infants.

The next phase in this interesting evolution of biochemical and clinical synthesis was the work of Lapin,[20] whose article in 1944 combined Anderson's investigation with his own clinical observations. Lapin described what he termed "metabolic diarrhœa" or the cœliac syndrome. There were five chronological phases: (i) occurring at birth, cystic pancreatic fibrosis; (ii) at three to twelve months of age, starch intolerance; (iii) at one to eight months of age, infantile steatorrhœa; (iv) at ten to sixty months of age, cœliac disease steatorrhœa; (v) the "allergic cœliac" syndrome, as found in intolerance to various foodstuffs. Such a classification is obviously most useful from the clinical point of view; but will become a handicap unless it is realized that every case of failure of an infant to thrive cannot be exactly fitted into one or other of its subdivisions. Indeed, Lapin's article includes a comprehensive list of the characteristics of each type referred to above, in accordance with the water content and other characteristics of the stools produced by the infant. Such a digression is useful in confirming clinical data when necessary, but would be too cumbersome for routine use. Robbin and Bernard[23] also have produced a table of characteristics of the various forms of stools corresponding to the different types of infantile "indigestion". This, too, is rather rigid.

Clinical Approach.

As in many other lines of advance in modern medicine, the old may readily be combined with the new, and thus a

check will be given to over-enthusiasm for the practical and theoretical investigations, which would otherwise outrun the bounds of practical medicine and lead on into a realm which would ultimately lose the touch of reality and thus become a hindrance rather than a help. The following tabulation of clinical approach, which is modified from Anderson and Lapin, is therefore offered as a basis for trial and as a practical solution for some of the difficult problems sometimes encountered in infants:

1. Under or over feeding.
2. Systemic infection; the onset sudden, with fever, and early subsidence.
3. (*a*) Acute infection and toxæmia; such as summer diarrhœa or nephritis; (*b*) following an acute infection—subacute or chronic phases.
4. Cœliac syndrome "crises"; cases not at first recognized as due to this syndrome.
5. Mesenteric glandular enlargements, such as may be seen in tuberculosis.
6. "Metabolic diarrhœa", due to (*a*) pancreatic fibrosis, (*b*) fat intolerance, (*c*) starch intolerance, (*d*) true cœliac disease, (*e*) allergic manifestations.

Ætiology

Perhaps it may be said that the cause of cœliac disease in childhood is either a toxic-infective process or may be due in part at least to vitamin *B* deficiency. It is tolerably clear that a theory of defective absorption from the bowel will not alone explain all cases of cœliac disease. For example, it is well known that bananas are well tolerated by these patients. This fruit contains 20% of sugar in the form known as "invert" sugar. Cane sugar, on the other hand, is not well absorbed, and this fact may be due to absence of the enzyme "invertase" in the bowel, or the factor of impaired absorption may be the more important. In this connexion it is interesting to note that "invert" sugar is a mixture of the monosaccharides, glucose and fructose, and is so called because fructose rotates polarized light more strongly to the left than glucose does to the right. Cane sugar, on the other hand, is strongly dextrorotatory in polarized light, whereas the resultant disaccharide mixture of the banana sugar is lævo-rotatory.

A possible endocrine factor in cœliac disease is not to be forgotten nor too much stressed. Herter, referred to earlier in this article, suggested the possibility of a pituitary factor, and Still, in 1918, reported good results with polyglandular preparations in some of his cases. A clinical similarity between Addison's disease and cœliac disease has been noted and is emphasized in the pronounced asthenia, lack of muscle tone and capricious appetite to be observed in both diseases. The gastro-intestinal signs are similar, and some pigmentation of the skin has been noted in severe cases of cœliac disease. As tetany may be present in cœliac disease, it has been suggested that the upset in calcium metabolism may be due, at least in part, to some abnormal functioning of the parathyreoid glands. Decalcification of bone may also be referred to, and this condition occurred in one of the patients to be mentioned later in this article.

Children suffering from cœliac disease frequently have a small liver. Normally in infants the liver is just palpable below the right costal margin, and in these cases the liver edge may not be defined. It may be recalled that some investigators hold that the bile salts unite with fatty acids in the bowel to form complex compounds, and as such most of the fat may be absorbed from the intestine. It has been asserted that this process is presided over by the adrenal secretions. In any event the term "acholia", at one time used in connexion with cœliac disease, sufficiently signifies its supposed linkage with liver function.

It may also be mentioned that, if we accept animal experiments as a guide to what occurs in the human body, then we can say that the fæcal fat bears no relation to the fat ingested in the food. This may rather upset our ideas of absorption of fat in cœliac disease. Sperry and Bloor[32] studied cats and dogs both fasting and receiving standard diets; they separated the fats into fractions and studied these fractions, with special reference to the source of the fatty material. The composition of the fæcal fat differed from that of the food fat. Cellular elements in the bowel in cœliac disease may account for some of the fat found to be present. Further, such substances may be present on account of vitamin deficiency, which is known to be present in some of the cases. There is also the question of infection, which is commonly present and is possibly related to vitamin *A* deficiency, as has been revealed in certain cases of the cœliac syndrome.

The question of vitamin *A* deficiency is of some interest in the cœliac syndrome. Moore,[33] in 1929, showed that carotin possessed an action similar to that of vitamin *A*. Ingestion of excess of carotin by albino rats leads to great excess of the vitamin *A* colour reaction, but to no increase of pigment in the liver. In 1938, Clausen[34] and McCord investigated the carotinoid content of the plasma, and concluded that large amounts of these substances passed to the fœtus via the placenta. Infection caused a prompt and considerable fall in the levels of carotinoids and of vitamin *A* in the plasma. This fall was in part due to low intake during the period of infection, and in part it resulted from the presence of fever. Flax and Barnes[35] found a relation between intestinal motility and vitamin *A* absorption in fibrocystic disease of the pancreas. In one of their studies a second sibling died forty-five and a half hours after birth; this suggested a congenital pancreatic fibrosis. However, in view of the work of Clausen and McCord, referred to above, deficient diet of the mother may have played a part in these cases.

More recently, in 1944, Luzatti and Hansen[20] investigated ten children with the cœliac syndrome; in eight of these cases the serum lipids were estimated. The iodine numbers of the fatty acids of all fractions of the lipid content of the serum of these eight patients were abnormally low. Luzatti and Hansen regarded the determination of the iodine numbers of the serum fatty acids as a possible means of ascertaining the ability of the bowel to absorb fat. They stated that deficient fat absorption in the cœliac syndrome seemed to be difficult to explain with our present knowledge of the physiology of fat metabolism.

Analysis of Thirty-Five Cases of Cœliac Disease.

During the period from 1933 to 1943, 35 cases of cœlic disease occurred at the Children's Hospital Melbourne. Male patients numbered 23 and female patients 12. The youngest patient was aged one year. The average age at onset of the disease was two years and eight months. Six patients died, three males and three females. The average age at death was four years and eight months. The causes of death were as follows: toxic hepatitis and marasmus, one case; unresolved bronchopneumonia (asthmatic bronchitis), one case; intercurrent pneumonia, one case; thrombocytopenic purpura, acute gastro-enteritis and hæmorrhage, one case; pyelonephritis, one case; urethral calculus and bronchopneumonia (following perineal section), one case. Of the 29 survivors 17 have been examined, six have sent replies from country districts on a prepared question form, four have failed to answer letters of request for information, one recently traced may reply, and one is untraced.

A detailed survey of all the 35 cases would reveal no new features in the clinical history of these patients. The two outstanding features were the weight charts, which showed the usual fluctuations, and the temperature recordings. In 15 of the 35 cases the temperature rose at some time during the illness to over 100° F. (37·8° C.), the maximum in any case being 105° F. (40·6° C.). Ten maintained a fairly steady temperature, and by that is meant that there was no continued rise suggestive of some complicating factor. In a further ten cases the temperature hardly varied from the normal, an occasional rise to 99° F. (37·3° C.) only being recorded. No radiological evidence of rickets was found; but in one case some bone rarefaction (poor calcification) in the arms and in the leg bones was reported. A diagnosis of scurvy was made in one case. There was no definite evidence of macrocytic anæmia in any of the patients of the series, but the blood of some had low hæmoglobin levels. Upper respiratory tract infection was not a prominent feature, nor was *otitis media*. Pneumonia was not common; but one child, who died at the age of two

years and one month from intercurrent pneumonia, probably had fibrocystic disease of the pancreas, and a very large thymus gland was present. The pancreas was not microscopically examined, but was reported to be very small and to have given an impression of atrophy or of imperfect development. One patient who has survived is a mental defective. Another suffered from the effects of poliomyelitis involving the lateral abdominal wall musculature, and thus the effect of abdominal distension was heightened; recovery of muscle tone in this case, which was not severe at any time, took place in the course of two months. Hypochlorhydria with mucus in the fasting gastric contents was present in one case. The stool fat content invariably diminished on commencement of treatment, whether this was by dietetic means alone or by the addition of vitamin preparations as in modern practice. Variations in the percentage of fat in the fæces occurred from time to time during the course of the illness, and subsequently reached normal levels. Splitting of stool fat always reached a high level, thus showing no significant abnormality.

Diagnosis.

The diagnosis offered some interesting problems. In more than one case abdominal distension suggested the presence of peritonitis, and tuberculous peritonitis was naturally called to mind. Actually one patient was admitted to hospital with this diagnosis. The shrunken, wizened appearance seen in a young patient with cœliac disease lends a strong prop to the idea of ascites. The intradermal Mantoux test was performed on many patients in the series. As cœliac disease is one of relatively long duration, and furthermore is subject to remissions during its course, all the conditions enumerated in the discussion on clinical approach may need to be considered in any case of illness in childhood in which diarrhœa is present. However, fibrocystic disease of the pancreas is probably the most important in the list on account of its bad prognosis. The one patient in this series who was earlier referred to as having a poorly developed or atrophic pancreas, presented some of the features of pancreatic fibrosis as an underlying cause of death. On the one hand, the child had the complicating features of pronounced thymic enlargement, as well as being over the age of one year (two years and two months at death); this is uncommon in this condition, which is usually fatal at or before the end of the first year of life. On the other hand, however, the child, a female, showed the typical features of cœliac disease, and there were other confirmatory facts in the clinical history. The important points with regard to a probable diagnosis of fibrocystic disease of the pancreas were the presence of a good appetite with continued wasting, and the supervening pulmonary disease, some form of which appears to be an integral part with the pancreatic disability in this variety of the cœliac syndrome.

Prognosis.

The death rate in cœliac disease as reported by various authors varies considerably, and the following are illustrative: Hardwicke (1923 to 1938), 30%; Parsons (1932), 10% to 12%; Neal (1935), 10% to 12%; present series (1933 to 1943), 17·1%.

Sheldon[qm] refers to various causes of death, such as sudden relapse of diarrhœa, progressive exhaustion and any intercurrent infection, such as measles or pneumonia, which are badly borne by there patients. In this series stress is laid on the fact of return to normal in regard to height in most cases, whereas the average weight for age has not quite been reached by most of the patients. With the exception of the mentally deficient child referred to earlier, mental development does not appear to have been seriously interfered with in most cases.

In the height-age chart (Figure I) the uninterrupted line represents the average height for age of the males, and the interrupted line the average height for age of the females. The triangles represent males and the crosses represent females. Seventeen children were examined, of whom there were 14 up to the age of 14 years, and three aged over 14 years. Of the 14 children up to the age of 14 years, two males and one female are definitely under average height. One female is only three-quarters of an inch under average (age 13 years). Of the three aged over 14 years (two males and one female), the female is aged 19 years and is over normal height and weight, dislikes fatty foods and is intolerant of eggs. One of the males, aged 17 years, is a little under average height and weight; his diet is normal and he stammers a little. The other male is aged 18 years;

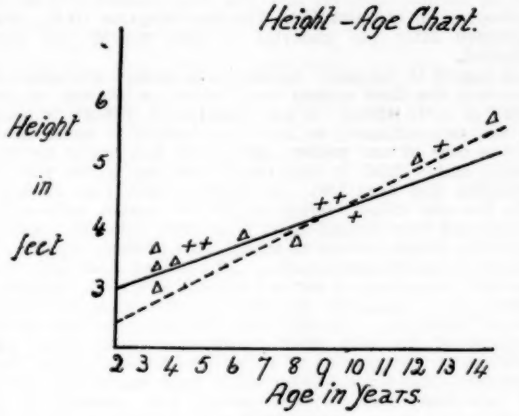

FIGURE I.

his diet is normal, but he is two pounds under weight. He had odd vomiting attacks up to the age of 14 years, and he suffers from enuresis.

In the weight-age chart (Figure II), the uninterrupted line represents the average weight for age in males, and the interrupted line the average weight for age in females. The triangles represent males and the crosses represent females. This chart illustrates the weights of 14 of the 17 patients interviewed—that is, those aged up to 14 years.

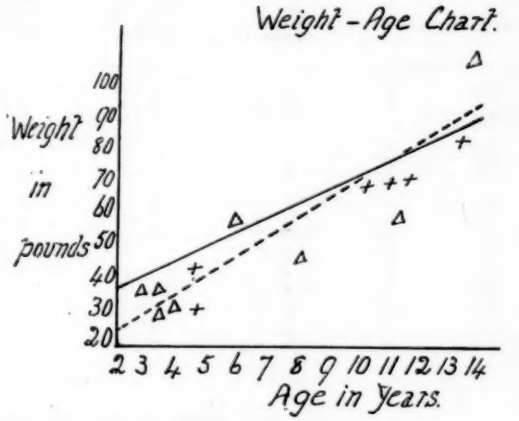

FIGURE II.

The other three were aged over 14 years, and were fully dealt with in Figure I. In contrast to the last, this chart shows that only two males and one female are over the average weight. The oldest male, aged 14 years, has caught up in both height and weight and exceeded the average for age. Of six who sent replies to questions from the country, one was able to come in and is mentioned in the previous data. Of the remaining five, one male is over average height (no weight given). Two males are over the average height and weight, and one male and one female are under average height and weight.

Table I summarizes the condition of the children when examined. It will be seen that some patients are still obliged to adhere to a diet, either rigid or modified in various ways, and that three were found to have abnormal prominence of the abdomen. Dental caries was fairly common; but this finding is so frequent in children generally in this country that no particular significance can be placed on it.

Table II summarizes the condition of six children who live in country districts and who were unable to come to Melbourne for examination. The one exception (G.M.) was examined after the material for this survey had been collected.

In regard to the older children, an attempt was made to ascertain the time missed from school on account of the result of their illness. It was found to be difficult to make an accurate estimate, as time was broken in some cases. In the case of one patient, B.C., who was under average height and weight, it was stated that one whole year of schooling had been lost. In another case, that of K.W., who was also under average height and weight, little or no school had been missed apart from time spent in hospital. As to the grade reached at school, no significant facts were elicited from this information. All children examined, with the one exception of the sub-normal girl, J.G., appeared bright and reasonably intelligent. The male D.F. is a mental defective. The girl I.C. was of some interest on account of the fact that ingestion of cane sugar was followed by the passage of large, pale "bubbly" stools. An endeavour was made to have blood sugar estimations made in this case; however, the patient was resident in the country and arrangements for her attendance in Melbourne could not be made.

Radiological Survey.

I am indebted to Dr. Colin Macdonald for the following statement of the relation between X-ray appearances of the bony skeleton and disordered calcium metabolism in cœliac disease: (i) delay in ossification and bony growth: (ii) transverse lines in the metaphyses; (iii) osteoporosis (decalcification); (iv) osteomalacic deformities; (v) cœliac rickets and deformities; (vi) dwarfism. Dr. Macdonald radiologically examined the long bones of 15 of these children, their age at the time of the examination ranging from three and a half years to nineteen years. Dr. Macdonald's findings were as follows. The osseous radiological evidence of cœliac disease was an expression of deficient calcium absorption, and in this respect was akin to the rickets of vitamin *D* deficiency. In cœliac disease the milder osseous manifestations were transverse lines in the metaphysis and shaft, delay in epiphyseal ossification and bony growth, and osteoporosis, and in severe cases cœliac rickets (with associated deformities) and dwarfism. All the 15 patients had their bones compared with those of normal children of the same age and sex. None showed delay in epiphyseal ossification or bony growth; four showed transverse lines in the metaphyses and shafts; one showed mild osteoporosis; but in none was there any suggestion of cœliac rickets or dwarfism. Dr. Macdonald considers that there is nothing diagnostically characteristic in the transverse lines so frequently seen in children's bones; they appear whenever there is a temporary impairment in the process of ossification, and this occurs whenever any disease process disturbs (even temporarily) calcium metabolism. The severe grades of osseous manifestation of cœliac disease frequently reported from European and American sources would therefore appear to be rare in Australia.

Treatment.

The graded diet used at the Children's Hospital, Melbourne, is as follows:

CŒLIAC DIET.

Stage I:

Boiled skimmed milk, 9 or 10 ounces; five to six feeds per day. Milk must be machine separated; otherwise use a dried skin milk powder. Vitamins: "Adexolin", strained orange, lemon or tomato juice, which may be sweetened with glucose. "Marmite" or wheatgerm may be given in milk feedings, two teaspoons per day.

TABLE I.
Seventeen Patients Examined.

Name.	Sex.	Age at Onset. (Years and Months.)	Age When Examined. (Years and Months.)	Duration of Hospital Treatment. (Months.)	General Condition.
K.G.	M.	1, 8	6, 5	3·75	Appears a normal child. Using number 3 cœliac diet.
J.G.	F.	1, 4	4, 9	5·0	Slightly reduced fat in diet. Mentally subnormal child.
E.K.	F.	4, 10	11	11·0	Using an almost fat-free diet. Two motions daily; fæces are "pale". Abdomen protuberant and soft.
W.H.	M.	1	3, 3	1·5	Abdomen a little protuberant, otherwise well.
F.D.	M.	1, 11	11	3·25	Abdomen a little protuberant, otherwise well.
I.C.	F.	2, 9	13	2·0	Eats all foods with plenty of fat. Avoids cane sugar, as stools become pale and frothy if it is taken.
R.G.	M.	3	8	6·0	Appears a normal child. Normal diet.
A.M.	M.	1, 3	3	1·25	Had been well, but suffering an attack of bronchitis when examined.
E.P.	M.	3, 5	18	7·0	Normal diet. Enuresis.
D.F.	M.	1, 6	3, 6	1·0	Had an upper respiratory tract infection when examined. Mental defective.
G.F.	M.	1, 11	4	1·0	Appears a normal child. Using a diet restricted in fat.
D.B.	F.	4, 7	9, 3	6·0	Appears a normal child.
S.B.	F.	4, 11	10, 7	1·75	Appears a normal child. Diet normal.
T.D.	F.	1, 4	4, 8	1·75	Appears a normal child.
D.R.	F.	9, 2	19	3·0	Appears normal young woman. Eggs disagree, and fatty foods are avoided.
T.S.	M.	1, 10	14	16·0	Appears a normal youth. Diet normal.
L.V.	M.	4, 4	17	4·0	Appears a normal youth.

TABLE II.
Patients Who Sent Replies from the Country and Who were not Examined.

Name.	Sex.	Age at Onset. (Years and Months.)	Age when Examined. (Years and Months.)	Duration of Hospital Treatment. (Months.)	General Condition.
R.H.	M.	1, 5	—	2	Diet normal. Over average height.
D.M.	M.	2, 1	—	4	Diet normal.
J.R.	M.	1, 10	—	1	Diet normal.
K.W.	M.	1, 8	—	14	Diet normal. Under average height and weight.
B.C.	F.	2, 9	—	5	Diet normal. Under average height and weight.
G.M.[1]	M.	1, 9	4, 5	3	Abdomen prominent and doughy. No fat in diet. Stool normal.

[1]This was the child subsequently brought from the country and examined.

Stage II:

6 a.m.: Boiled skimmed milk, 10 ounces.

Breakfast: "Weeties" (small helping) with wheatgerm, two teaspoons. Rusk or "Uneeda" biscuit with "Marmite". Boiled skimmed milk, 10 ounces.

11 a.m.: Orange, lemon or tomato juice (strained), three ounces. Glucose, 0·5 to 1·0 ounce. Water, two or three ounces.

Dinner: Fish (steamed), rabbit, minced or scraped lean meat or liver. A little sieved vegetable—marrow, pumpkin, cauliflower, tomato or cabbage. Well ripened banana, mashed. Skimmed milk, either as jelly or to drink.

Tea: Fish, rabbit or scraped meat as at midday. Rusk or "Uneeda" biscuit with "Marmite". Mashed ripe banana. Boiled skimmed milk, eight ounces.

The amount of well-ripened banana may be increased if the child is tolerating it. Sieved stewed fruit may be given.

Stage III:

6 a.m.: Boiled skimmed milk, 10 ounces.

Breakfast: "Weeties" (small helping), with wheatgerm, two teaspoons. Rusk or "Uneeda" biscuit or crisp dry toast with "Marmite". Lean crisp bacon, half an ounce. Boiled skimmed milk, 10 ounces.

11 a.m.: Orange, lemon or tomato juice (strained), three ounces. Glucose, 0·5 to 1·0 ounce. Water, two or three ounces.

Dinner: Fish (steamed), rabbit, minced or scraped lean meat or liver. A little mashed potato (well cooked). A little sieved vegetable. Mashed ripe banana or sieved stewed fruit, such as apple, apricot or peach. Skimmed milk jelly or boiled skimmed milk to drink.

Tea: Fish, *et cetera*, as at dinner. Mashed ripe banana. Rusk or crisp toast with "Marmite". Boiled skimmed milk, eight ounces.

Next, butter (about five grammes) may be introduced, and later a little milk fat. The introduction of fat must be done very gradually and the child's condition carefully watched.

When, following the work of May, McCreary and Blackfan,[37] vitamin injections were used, the dietary plan was (and still is) retained during hospital treatment and afterwards in the out-patient department. May, McCreary and Blackfan[37] employed a normal diet for their patients after the first few weeks of treatment.

The course of daily intramuscular injections employed in Melbourne extends over three weeks as follows:

First day: liver extract (Parke, Davis and Company), two millilitres.

Second day: "Vibex" (50 milligrammes per millilitre), two millilitres.

Third day: liver extract, two millilitres.

Fourth day: nicotinic acid (50 milligrammes per millilitre), two millilitres.

Fifth day: liver extract, two millilitres.

This is repeated until 21 injections have been given. After a spell of six weeks, the course of injections is repeated. Clinical improvement is judged by improvement in the general condition, and by the character and the results of fat estimations of the fæces as well as by the weight. Weight charts are kept for all patients. A full ration of orange juice is provided for all children, and this consists of a maximum of two ounces per day. In addition "Adexolin" is administered up to 20 minims per day according to age. Fresh air and sunlight, regard being paid to weather conditions, have been found to be useful adjuvants to treatment.

Summary.

1. A short historical survey of cœliac disease is given.
2. Modern trends in investigational methods are outlined.
3. Some problems in relation to the disease are discussed.
4. Reference is made to the question of ætiology.
5. Thirty-five cases of the disease are analysed, and the problem of diagnosis is considered, together with the prognosis.
6. The results of a radiological survey are submitted.
7. Treatment is briefly discussed.

Acknowledgements.

I wish to thank members of the honorary medical staff of the Children's Hospital, Melbourne, for permission to investigate patients under their care. My thanks are due particularly to Dr. J. W. Grieve for his constant help and advice during the course of this study. Dr. Colin Macdonald, honorary radiologist to the hospital, has kindly discussed the radiological findings with me, and submitted his opinion thereon. The almoner to the hospital, Miss Hodge, and her staff have been most helpful at all times.

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Reviews.

THE ENDOCRINOLOGY OF WOMAN.

As the author states in the preface, "Endocrinology of Woman" has been prepared under the very trying conditions of war, but nevertheless the consequent difficulties appear to have been successfully overcome, as both the subject matter and the production are excellent.[1]

[1] "Endocrinology of Woman", by E. C. Hamblen, B.S., M.D., F.A.C.S.; 1945. Springfield: Charles C. Thomas. 10″ × 6¼″, pp. 571, with many illustrations. Price: $8.00, post paid.

The book is divided into five parts. In Part I each chapter commences with a summary of the history of important discoveries in relation to the various endocrine glands. The embryological development and anomalies, the anatomy, histology and secretory control, the chemistry, physiology and interrelationship with the other ductless glands are then described in connexion with each gland. Although the book is entitled "Endocrinology of Woman", there is a chapter on the testis, because, as the author states, such a study is necessary for the understanding of the problems of intersexuality, female androgen therapy, syndromes associated with virilization and sterility.

Part II is concerned with "applied" endocrine physiology. The effect of the action of the endocrine glands upon the development of the various functions at different stages of life is traced in this section. Ante-natal development, the changes in childhood and puberty, sexual maturity and finally sexual regression are described.

In Part III the methods used in the diagnosis of endocrine disorders are described. The importance of a complete history for which there is no substitute is stressed. Endometrial biopsy, histopathological and bacteriological methods, biochemical and metabolic studies are dealt with, as well as hormonal and radiological data.

The fourth part is concerned with functional disorders of the endocrine glands and contains a description of the various diseases of endocrine origin such as acromegaly, myxœdema and adrenal virilism.

In Part V the application of endocrinology to gynæcological disease is described. The sections dealing with abnormal uterine bleeding, sterility and the climacteric are of great value to the clinician, and the last chapter describing commercial endocrine preparations will prove most useful for reference.

As can be seen by this summary of its contents, the book covers a wide range, and it will prove of great value to all members of the medical profession because the author is a man of vast experience who has presented his views in a sound fashion, and has stressed the limitations of endocrine therapy. Finally the value of the book is enhanced by the bibliography, which comprises footnotes on each page and a list of references (all in English) at the end of each chapter.

PROGRESS IN RADIOLOGY.

"THE 1945 YEAR BOOK OF RADIOLOGY" has been received for review.[1] The diagnostic section is edited by Charles A. Waters and Whitmer B. Firor, while the therapeutic section is edited by Ira L. Kaplan. The book is published by the Year Book Publishers of Chicago. This work has been published now for many years, and the present edition is well up to standard. It consists of reviews of current radiological literature. The chapters are methodically arranged and the illustrations are wonderfully good. The articles on infantile bone diseases (rickets, scurvy *et cetera*) and infantile deformities by R. S. Bromer and J. F. Brailsford are particularly good and are well illustrated. Many of the conditions described are exceedingly rare and the work shows rather a tendency to deal with such subjects. March fractures, including fractures from carrying heavy shoulder loads, are described and illustrated, and a very good case of *leontiasis ossium* is reported in a child aged twelve years. High altitude joint pains (bends) are discussed by S. F. Thomas and Owen L. Williams, and they have demonstrated gas collections about affected bones in the soft tissues at heights of 20,000 feet and upwards. These collections are absorbed gradually as the patient descends to earth level. Intervertebral disk protrusion is mentioned, and this may be multiple. Myelography is recommended before operation. Unusual conditions are reported in the various systems such as Hodgkin's disease of the chest, thymomata, dermoids *et cetera*. A good deal of space is devoted to the cardiovascular system and the use of opaque media for demonstrating various arterial and venous conditions. Waldron Smithers writes on the short œsophagus (thoracic stomach) and its association with peptic ulcer. Reports suggest that thiamin hydrochloride in ten-milligramme doses by mouth three times daily before meals may be of use in the relief of cardiospasm. "Priodax" has apparently supplanted the earlier preparations in gall-bladder investigation. The use of this phenolphthalein-free drug does away with the nausea and diarrhœa so often met with. A detailed description of myelography with "Pantopaque" is given in the chapter on the nervous system and some excellent illustrations of prolapsed disk are provided.

Little new work is described in the section on technique. In the therapy section attention is drawn to the difficulties encountered in all parts of the world owing to shortage of supplies and of assistant staffs, and so there is little new to report, although much heavy routine work has been carried out. The opinion is expressed that it would be well for radiotherapy if the Australian rule was followed in supplying radium or radon only when specialists are available to see to its proper use. Attention is drawn (Brailsford) to the danger of biopsy in malignant disease of bone, as there is danger of disseminating the tumour cells. The view is stressed that only trained specialists should be allowed to practise radiotherapy. No new ground is covered in the descriptions of therapeutic technique.

The year book should have a place in the library of every radiological specialist.

[1] "The 1945 Year Book of Radiology"; Diagnosis: edited by Charles A. Waters, M.D., associate editor, Whitmer B. Firor, M.D.; Therapeutics: edited by Ira L. Kaplan, B.Sc., M.D.; 1945. Chicago: The Year Book Publishers, Incorporated. 9" x 6", pp. 464, with many illustrations. Price: $5.00.

OBSTETRICS AND GYNÆCOLOGY.

No synopsis of any medical subject can claim to be a complete textbook on that subject, but "Synopsis of Obstetrics and Gynæcology", by Aleck W. Bourne, is certainly as complete as any synopsis could be.[1] Most of the subject matter is set out in tabular form, and this enables a quick revision to be made, embodying the salient points of each specific condition. For this reason the book is of value both to students and to medical men who are doing a refresher or a post-graduate course. This is the ninth edition of a well-known book, the popularity of which never wanes. Perhaps its greatest virtue is the dogmatic manner in which all subjects are treated; there are no "ifs" or "buts" and the author does not hesitate to state what treatment he uses for any condition, and does not deliberate over controversial matters. He states facts which are backed by his own practical experience, the extent of which is well known, and this makes the book all the more valuable from a student's viewpoint.

The normal and abnormal events and the complications which occur during pregnancy, parturition and puerperium are all very well described, and the sketches illustrating the various procedures, while being rather on the small side, are clearly drawn and adequately illustrate their subject. In the section on gynæcology, the various diseases are concisely described, and the operative methods of cure, whilst being briefly dealt with, nevertheless comprise the main points. The treatment of bacterial infections by both sulphonamide and penicillin therapy is discussed. A valuable chapter covers the relative merits of radium, deep X-ray therapy and surgery in the treatment of pelvic malignant disease. To mention every subject covered is not possible, but, despite the necessary brevity of the book, it can be recommended to all who need such a synopsis.

RADIOLOGICAL DIAGNOSIS OF OSSEOUS LESIONS.

"THE OSSEOUS SYSTEM: A HANDBOOK OF ROENTGEN DIAGNOSIS", by Vincent W. Archer, Professor of Roentgenology, University of Virginia, has been received from the Year Book Publishers Incorporated, of Chicago.[2] As stated in the preface, it is dedicated to the occasional radiologist, and it can certainly be recommended for such use. The author gives a very short discourse on the general principles of interpretation; this section could be extended with advantage. Various regions are considered and illustrated, and stress is laid on the normal before reference is made to the abnormal conditions of bones. The pathological lesions of bones are covered shortly and concisely and are well illustrated. The illustrations are excellent and are produced in the "negative", so that the reader sees the illustration as it appears on the original film.

This book should be of great use to country practitioners (with small X-ray outfits) who are unable to consult a specialist on the subject.

[1] "Synopsis of Obstetrics and Gynæcology", by Aleck W. Bourne, M.A., M.B., B.Ch. (Cambridge), F.R.C.S. (England), F.R.C.O.G.; Ninth Edition: 1945. Bristol: John Wright and Sons Limited. London: Simpkin Marshall (1941) Limited. 7½" x 5", pp. 507, with illustrations. Price: 21s. net.

[2] "The Osseous System: A Handbook of Roentgen Diagnosis", by Vincent W. Archer, M.D.; 1945. Chicago: The Year Book Publishers Incorporated. 8" x 5½", pp. 320, with many illustrations. Price: $5.50. Australian, 40s.

The Medical Journal of Australia

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All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

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THE PARENTERAL USE OF VITAMINS.

VITAMINS, like other foods, should be taken by mouth. They should form part of the food that is eaten every day; a healthy person taking a balanced diet should have no need to take any of the concentrated vitamin preparations that are to be found in such numbers on the market. If an individual has undergone a period of dietary deprivation or imbalance, if he has suffered from certain types of illness, he may stand in need of vitamins and this need may sometimes be met by the oral administration of one or more vitamin preparations. A great deal of unnecessary self-medication with vitamins is prevalent. Non-medical persons are too ready to assume that a feeling of well-being is due to the ingestion of a proprietary vitamin preparation recommended by a neighbour, and they recommend the preparation to others who are quite certain that it will benefit them too. People who should know better are guilty of this kind of thing and it may be justifiable to wonder whether practitioners who include vitamin preparations among their stock remedies are always careful about the indications for their therapy. When we come to the parenteral use of vitamin preparations, we come to a realm of therapeutics in which there is a great deal of confusion. Estimates have been made and lists have been drawn up showing the quantities of vitamins that should be consumed by normal persons; but there is no recognized standard for vitamins parenterally administered. What has to be determined is how much of a vitamin injected subcutaneously or intravenously is likely to be effective, what preparations should be used and when. Clearly the injection of vitamins may become, and we believe sometimes does become, a lucrative method of treatment for an unscrupulous practitioner whose preference for this method seems to appeal to a gullible clientele.

An attempt has been made to bring some order into this confused subject by Franz J. Ingelfinger, who is assistant professor of medicine at the Boston University School of Medicine.[1] He lists the patients for whom parenteral administration of vitamins is justified as follows: those who are unable to take any food by mouth; those suffering from a defect in intestinal absorption; those who are so acutely deficient in vitamins that the delay of intestinal absorption must be circumvented; those who require fairly large doses of vitamins, but suffer from a disorder that orally administered preparations might aggravate; those unable or unwilling to ingest a specific vitamin preparation that is indicated. The matter is not simple. The clinician cannot say to himself: "Mr. So-and-So needs every day such-and-such a quantity of vitamin B_1; I shall inject that amount and all will be well." The vitamin injected is not all effective; and many factors, such as the patient's general condition and his Calorie intake, must be considered. The patient's vitamin reserve can be tested in the laboratory; when this cannot be done an estimate must be made. In this estimation the patient's whole nutrition must be considered as a unit. The vitamin and caloric intakes cannot be considered separately, and intensive therapy with a single vitamin is not judicious in the average case. It is interesting to note that a man completely starved does not develop an acute vitamin deficiency. If, however, he terminates his fast by consuming large quantities of sugar, signs and symptoms of vitamin deficiency may develop rapidly. This is because vitamins take an active part in metabolic processes. Also, a patient who has had a calorically deficient intake may have better vitamin stores than one who has taken a diet calorically adequate but deficient in vitamins. It follows further that a man subsisting on parenteral nutrition is more in need of vitamins if his caloric intake is adequate than if it is deficient. By far the greater part of evidence indicates that parenteral vitamin injections that are given for the purpose of maintaining nutrition should never consist of a single vitamin. Only when a specific indication for treatment with a single vitamin exists, as in the vitamin *K* deficiency of obstructive jaundice, is the injection of one particular vitamin warranted.

Thiamine chloride (vitamin B_1) is present in the blood both as free thiamine and as a phosphorylated compound, diphosphothiamine. The latter, also known as cocarboxylase, appears to be the physiologically active form of vitamin B_1 and is found in the tissues and cellular elements of the blood. Free thiamine, which constitutes only a small fraction of the blood's total thiamine content, is present chiefly in plasma. The concentration of thiamine in the tissues of the body is about ten to twenty times that of whole blood; the total vitamin B_1 content of a normal man averages about 25 milligrammes. When illness and poor nutrition occur the tissue stores of thiamine may be heavily reduced. When solutions containing pure thiamine are given by subcutaneous or intravenous injection, the vitamin is apparently phosphorylated rapidly in such organs as the kidneys and liver and is in part used in metabolic processes. Some is stored in the tissues, some is excreted in the urine and insignificant amounts are lost in the fæces and sweat. The amount that can be measured as having been stored and lost is extremely small; this means that a considerable amount of thiamine undergoes processes of metabolism or destruction that cannot be followed at present. Passing over some interesting dis-

[1] *The New England Journal of Medicine*, September 27 and October 4, 1945.

cussion by Ingelfinger on the determination of a patient's nutrition in regard to thiamine, we note that it is theoretically possible that the urinary excretion of thiamine is slightly less after subcutaneous administration or after a slow intravenous infusion than after a dose given intravenously during the course of half a minute. In spite of this, caution must be exercised in estimating the effectiveness of the addition of thiamine to a solution given by the intravenous route. It has been shown that fractions of the vitamin *B* complex given in this way may be washed out by diuresis. The minimal thiamine requirements of a normal adult person have been estimated to vary from 0·2 to 0·6 milligramme per thousand Calories. No benefit is said to follow the ingestion of more than one milligramme for every thousand Calories. Ingelfinger thinks, however, that if one is guided by the more generous estimates of the needs of a normal man, a patient who receives 1,500 Calories a day, whether by mouth or vein, should get about one milligramme of vitamin B_1 daily. Disease may or may not alter the thiamine requirement. It is safest to consider that the needs for thiamine are increased in disease and that a requirement of one milligramme per thousand Calories should be arbitrarily chosen. An allowance being made for a 25% loss in the urine, a patient unable to digest food but with presumably normal stores of thiamine (a patient, for example, with acute appendicitis and peritonitis) should receive parenterally three milligrammes of vitamin B_1 a day if he is given 2,000 Calories a day by intravenous infusions. Most patients who require parenteral administration of thiamine do not have normal stores of the vitamin. Of all the vitamins, thiamine chloride is the least easily stored by the body. Unless facilities are available for measuring the thiamine content of urine, the estimation of a patient's thiamine stores is practically impossible. The probable range that is proposed cannot be traversed. It may be stated, however, that the man with no signs of thiamine deficiency, but whose stores have presumably been depleted during his illness, should have his needs met if he receives ten milligrammes a day for ten days, after which the dosage may be decreased to the amount estimated as the daily requirement. Two facts in regard to thiamine need to be mentioned. The first is that sensitization to the vitamin may occur. The other is that biosynthesis of the vitamin occurs in the human intestine; this has been the subject of comment in these columns. Ingelfinger quotes authorities in support of the view that its implication in human nutrition is debatable.

Ingelfinger's discussion on thiamine has been described by no means fully, but at some length, first of all to show how the subject of parenteral administration of vitamins should be approached, and secondly, to give an indication of the complexity of the subject. But we must follow him a little further. Riboflavin, a water-soluble fraction of the vitamin *B* complex, is present chiefly in the red cells, but also in the tissues. The riboflavin stored in man's bodily tissues is four to six times greater in amount than his thiamine stores. Pure riboflavin may be injected subcutaneously or intravenously. It is apparently retained according to the needs of the patient when given parenterally in small doses, but when larger doses (five milligrammes or more) are injected, the urinary loss "is also affected by renal function, by the extent to which the blood stream is loaded and by other variable factors". The body's stores of riboflavin are less rapidly depleted than those of thiamine. From figures which Ingelfinger gives, it appears that a patient who has been ill or who has had a restricted riboflavin intake for less than two weeks requires no more riboflavin parenterally than that given to cover his daily requirement. For persons who have been subjected to possible riboflavin deprivation for longer periods, ten milligrammes for five days should, in the ordinary course of events, compensate for any depletion of stores that may exist. Niacin or nicotinic acid, another water-soluble member of the vitamin *B* complex, may be given parenterally. The amide (nicotinamide, niacin amide) should be used in order to avoid certain flushing reactions. When nicotinamide is given in large doses intravenously or intramuscularly, a rise in the total niacin content of the blood occurs, but the rise and its duration are small in proportion to the dose given. The metabolic fate of most of the injected nicotinamide is unknown; a small proportion may be recovered from the urine and some is probably methylated in the liver. It does not appear possible to saturate man with nicotinamide, as can be done with vitamin B_1 or riboflavin. The daily ingestion of large doses increases the output of the vitamin only moderately or not at all. The reason is not clear. Either nicotinamide is stored or destroyed in the body, no matter how much is given in excess of the body's needs, or all its derivatives appearing in the urine have not been identified. Regardless of the size of the dose or the route by which it is given, it appears that less than 20% and often not more than 10% is excreted in the urine. By analogy with other vitamins it is assumed that the requirement for niacin is roughly parallel to the total metabolism. Thus six milligrammes of niacin for each thousand Calories is the dose recommended for the normal person. For a person who is ill Ingelfinger thinks that ten milligrammes may be suggested as an adequate dose for parenteral administration, although, as he points out, the influence of fever, starvation and thyreotoxicosis on man's niacin requirement is totally unknown. Biosynthesis of nicotinamide is not only held to occur in the human intestine, but it is also believed to contribute a substantial part to the nicotinamide requirement. At the risk of our prolonging this account unduly, other members of the vitamin *B* complex must be mentioned. Two other water-soluble members of the vitamin *B* complex have been identified and studied—pyridoxin and pantothenic acid. It has not been established unequivocally that these are essential to man and the part, if any, played by them in human nutrition has not been defined. In spite of this uncertainty, Ingelfinger states that there is little doubt that vitamin *B* complex contains factors essential to man's health. On the basis of annual experiments vitamin-like activity has been claimed for many substances. Most of them are untried entities in human nutrition. It should be noted, however, that R. J. Williams, writing in "The Annual Review of Biochemistry" for 1943, states that studies dealing with distribution (which he names) have indicated that the vitamins thiamine, riboflavin, nicotinic acid, pantothenic acid, pyridoxin, biotin, inositol and folic acid are universally distributed in all living matter. He admits that their biological functioning is not fully known. In order to supply vitamin *B* complex parenterally liver extract must be used. Ingelfinger mentions some important work by Clark on the virtue of concentrated as opposed

to crude liver extracts. He also points out that no definite indications for parenteral vitamin *B* complex therapy can be given.

Having mentioned Ingelfinger's observations on thiamine, riboflavin, niacin and other members of the vitamin *B* complex, we must forbear to deal with his discussion on vitamins *C*, *A*, *D*, *E* and *K*. The members of the *B* complex that have been mentioned form a group that should be studied together, as they should probably be used together in treatment. Ingelfinger has produced an extremely valuable review; it is clear and well documented. His message, which must be plain to every reader, may be reinforced by short reference to "a concept of deficiency states" advanced by H. D. Kruse.[1] Kruse holds that, while each avitaminosis shows a specificity of the particular tissue sites in which characteristic lesions appear, the changes in each tissue may be quite different, depending on the velocity at which they have developed, whether they are "acute" or "chronic". Each of these categories may be further divided into severe or mild groups. Thus deficiency conditions may be severe acute, severe chronic, mild acute and mild chronic. Kruse holds that the rate at which the tissue process develops determines the maximum rate at which any curative force can act. Most of the avitaminoses produced experimentally in laboratory animals have been of this acute type and clinicians have correspondingly come to expect vitamin therapy to produce responses with dramatic rapidity. Cases of human riboflavin deficiency so far recognized in the clinic have been almost exclusively acute. This is partly because the rapidly appearing tissue changes and their related symptoms are more apt to attract attention than the insidious slow chronic alterations, but partly also because specific therapy in the latter type acts with such disappointing slowness as to be dismissed by the clinician as unsuccessful. Following recent surveys and the use of new diagnostic methods, Kruse holds that chronic malnutrition must be much commoner than acute. He also believes that in the past in many recognized cases of avitaminosis treatment has been incomplete, intensive therapy having been merely continued until the acute symptoms subsided or until the level of vitamin in the blood became normal, while the underlying chronic condition of the affected tissues was only partially reversed.

[1] Quoted by C. S. Lanford and H. C. Sherman in "The Annual Review of Biochemistry", Volume XII, 1943, page 420.

Current Comment.

EPISTAXIS.

EVERY healthy schoolboy sooner or later has the disturbing experience of a bleeding nose; he early discovers that some of his compeers exhibit this phenomenon on slight nasal impact, whereas others can take considerable punishment without external manifestation. The liberal hæmorrhage at amateur boxing contests will also draw attention to the susceptibility of the facial prominence to shed its "ruddy drops". In general the traumatic epistaxis of sporting origin does not call for surgical treatment, as Nature soon seals the internal wound and a damp sponge restores the face to normality. Very different is the spontaneous epistaxis arising from diabetes, hæmophilia, leuchæmia, streptococcal infection and that associated with hypertension in elderly people; then there are those cases caused by permanent damage resulting from trauma in earlier life. Henry H. Amsden insists that epistaxis is in the province of the surgeon and gives a list of measures which can be adopted to arrest the bleeding.[1] Cauterization of the offending mucous membrane is the most efficacious method when less complex devices fail and the bleeding point is accessible, but various other expedients have been advocated such as simple pressure, pressure with pads soaked with hydrogen peroxide or adrenaline, post-nasal plugging, submucous resection, and the application of various hæmostatic agents such as a crystal of chromic acid, fresh tonsillar tissue, snake venom and even a piece of salt pork! The heroic resort to ligature of the external carotid has actually been considered necessary in special cases. The author omits the psychic assuagement elicited by a large household key being applied to the back of the neck—one assumes that the Yale lock is rendering this obsolete.

Discussions on epistaxis in medical literature make little reference to the problem why hæmorrhage should so easily arise from the nasal cavity. Bleeding into the middle ear or a cranial sinus is fortunately rare; the loss of blood from bruised pinna, lip or cheek is usually trivial. What is the causative factor in this readiness of the nasal mucous membrane to lose blood? Anatomical inquiry tells us that the mucous membrane in the sinuses and meatuses and on the floor of the nasal cavity is very thin, but is thick and highly vascular on the septum and on the conchæ. The arrangement of the blood vessels is certainly remarkable. Many anastomosing arteries form a plexiform network beneath and in the substance of the mucous membrane; veins are disposed on a closely meshed cavernous plexus, whilst arterio-venous unions present, as Macalister was one of the first to observe, the character of erectile tissue. The simple explanation of this extreme vascularization is that one major function of the nose is to pre-heat the inspired air, and a vigorous bloodflow is therefore necessary; another function is to de-dust the inspired air. J. S. Haldane discovered that the air in a crowded theatre or concert hall soon becomes almost bacteria-free, as all suspended matter is trapped in the noses of the audience. It has been remarked that two races have the largest noses: the Nordic for pre-heating and the Semitic for de-dusting. Most spontaneous hæmorrhages arise from the Kisselbach area, an arterio-venous plexus on the antero-inferior portion of the cartilaginous septum. The region of greatest vascularization is the anastomosis between the spheno-palatine and the greater palatine arteries. When cauterization is difficult Amsden recommends for intractable cases submucous elevation of the tissue on the floor of the nose and lower meatus under "Novocain" anæsthesia.

THE EFFECT OF EMETINE ON THE HEART.

AMŒBIASIS is, of course, not only a disease of the tropics, as the well-known epidemics of the past in temperate climates have shown. Now that more active and latent types of amœbic infection are likely to be seen in Australia in soldiers who have served in endemic areas, it is of wider diagnostic and therapeutic interest. Emetine in one form or another is still the most valuable specific drug we have in this condition. It is known to have a toxic action on the myocardium in laboratory animals, but how common such an effect is in human subjects receiving a therapeutic dose, is doubtful. General opinion is that harmful effects on the human heart are rare. J. D. Cottrell and G. W. Hayward have recorded their investigations in a New Zealand general hospital which served with the Central Mediterranean Force.[2] They note that Heilig and Visveswar described electrocardiographic changes in man, including lowering and occasional inversion of the *T* waves. In their own inquiry they studied the heart rate, blood pressure and electrocardiogram of patients under treatment for amœbiasis. These patients were otherwise healthy men of age between twenty and forty years and received ten to twelve daily

[1] *The New England Journal of Medicine*, January 17, 1946.
[2] *British Heart Journal*, October, 1945.

doses of one grain of emetine, and quinoxyl retention enemata, followed by "Carbarsone" 0·25 gramme twice a day for eight days, then emetine bismuth iodide three grains at night for ten nights. They remained in bed while having emetine in any form. Electrocardiographic studies were limited by the short supply of sensitized paper, but 32 patients were studied before and after the giving of emetine injections, and eight before and after emetine bismuth iodide. The rate of return to normal when alteration of the electrocardiogram was found was followed in eight cases also. The findings after emetine showed occasional minor changes in the *P*, *P-R* and *Q* features of the tracings, but almost constant lowering of the *T* wave in one or more leads. Six out of twenty-one patients showed fall of blood pressure from 15 to 20 millimetres in amount; this was transient only during the middle of the course, and by the end of the course the blood pressure was normal. No changes were observed in the heart rate. After the administration of emetine bismuth iodide the electrocardiographic changes were of similar type to those after injected emetine, but no alteration occurred in the heart rate or blood pressure. In every case any changes which had been observed completely disappeared in eight to twelve days, leaving no evidence of myocardial affection. The authors state that both in this series and in 250 patients in their hospital under treatment for amœbiasis they have seen no evidence of myocardial insufficiency. They point out that none of their patients suffered from malnutrition, and that none showed any evidence of previous myocardial disease. Brigadier Bedford, Consulting Physician to Middle East Forces, has stated to the authors in a personal communication that he has seen only one certain and one doubtful case of toxic action of emetine on the heart in that command up to the end of 1944. This work confirms the previous belief that while emetine has a depressant effect on the heart, serious damage is extremely rare, and a negligible risk under conditions of proper care.

MUSCULAR LESIONS IN RHEUMATOID ARTHRITIS.

THERE is recent evidence of an awaking of interest in the chronic rheumatic diseases. *Arthritis deformans*, or rheumatoid arthritis, is still a baffling disease, and unfortunately a crippling one also. It is crippling not simply because it is recurrent, as the relatively benign though uncommon syndrome described as palindromic rheumatism shows. Further there is ample evidence that its unhappy joint manifestations are a destructive feature of what is undoubtedly a systemic disease. The febrile episodes, the enlargement of the lymph nodes and the spleen in the so-called Still's disease of childhood, the curious remissions that may follow icterus, the nutritional changes in the skin and peripheral circulation, and the strangely rapid and excessive muscular wasting all point in the same direction. This last-named change is the subject of some recent work by G. Steiner, H. Freund, B. Leichtentritt and M. E. Maun.[1] Some of these authors have collaborated in previous work on multiple nodular inflammatory lesions in peripheral nerves in cases of rheumatoid arthritis, and they have collected further evidence of the co-existence of a nodular neuritis. They have in the present article described the histological changes in muscles, obtaining their material by biopsies from eight patients suffering from rheumatoid arthritis, and in addition have in one case thoroughly examined useless and deformed lower limbs amputated because of gross distortion and immobility. Since pathological changes in skeletal muscle have often been observed in cases of systemic disease, elaborate controls were necessary. Muscle tissue from 196 random cases was examined for purposes of comparison. Full clinical notes of the arthritic cases are given, together with the anatomical findings. Degenerative changes were found, but in addition definite inflammatory lesions were discovered. These were nodular in type and were infiltrated with lymphocytes and plasma cells, though polymorphonuclear cells were rare. In the larger nodules epitheloid cells were found, and a striking feature was the increase in collagen bundles. The pathological lesion was a nodular polymyositis with secondary atrophy. Taking in conjunction the previously observed changes in the peripheral nerves, the authors classify the lesions as nodular neuromyositis. What is more interesting is that they think the changes are specific. This statement is not so easy to prove, but it is pointed out that the changes were identical in every case, that the nervous, synovial and muscular lesions had the same pattern, and that they were widely different from the changes seen in the characteristic nodules of fibrositis. Further, the lesions were widely scattered and not found merely in the neighbourhood of affected joints. They did not resemble the lesions of muscles in degenerative or inflammatory diseases so far as the authors' researches were pursued. One interesting finding in this work is that these nodules were found in cases in which the disease seemed to be inactive and "burnt out". This is not surprising when we consider the long history of some of the patients with rheumatoid arthritis. Even immobility of joints does not necessarily mean inactivity of the disease. It is unfortunate that specific therapy usually lags behind discoveries in pathology, but treatment is not only specific. Rheumatoid arthritis is a disease process whose apt handling demands as full an insight into the workings of the patient's body and mind as possible, and any additional knowledge is welcome.

[1] *The American Journal of Pathology*, January, 1946.

THE BRONCHO-PULMONARY SEGMENTS IN SKIAGRAMS OF THE CHEST.

THE anatomy of the bronchi distal to the main lobar bronchi and of the parts of the lungs related to them has until lately received only sporadic study. Physicians, radiologists and surgeons, even those interested in diseases of the lungs, have no doubt found the published accounts, illustrated with complex diagrams, far from easy to follow. In view of the known variability in the anatomy of the smaller bronchi and in the absence of indications that the study of the broncho-pulmonary segments, that is to say, of the portions of the lungs served by the principal branches of the lobar bronchi, was of much practical value, it is hardly surprising that interest in these structures has not been more general. In 1937 we published a paper of much intrinsic and historical interest entitled "The Broncho-Pulmonary Segments", by J. Hardie Neil, W. Gilmour and F. J. Gwynne, of Auckland, New Zealand, one of the first products of the revival of learning in this direction, in which was made one of the first of the attempts of recent years to evolve a segmental conception of pulmonary anatomy and to indicate some of the practical applications of such a conception.[1] The importance of this work was emphasized in this country in 1943 by G. Griffiths, who demonstrated material prepared by Neil at a clinical meeting in Sydney.[2] There has recently appeared an exhaustive account of the anatomy and radiology of the broncho-pulmonary segments, copiously illustrated, in which the subject is so clearly and systematically set out as to appear for the first time not at all complex but very simple. The account is contributed to a symposium to commemorate the fiftieth anniversary of the discovery of the X rays by W. K. Röntgen, and its authors are A. F. Foster-Carter and C. Hoyle, of the Brompton Hospital, London.[3] We commend it to the careful study of all who are concerned with the diagnosis or treatment of diseases of the lungs.

Foster-Carter and Hoyle have demonstrated characteristic radiographic appearances associated with the consolidation and with the deflation of each of the major subdivisions of the pulmonary lobes. Most readers who are experienced in the radiology of the lungs, on seeing the illustrations of Foster-Carter and Hoyle, will recognize that these appear-

[1] THE MEDICAL JOURNAL OF AUSTRALIA, July 31, 1937.
[2] THE MEDICAL JOURNAL OF AUSTRALIA, October 16, 1943.
[3] *Diseases of the Chest*, November-December, 1945.

ances are familiar to them, but that they have not previously grasped their full import. It is stated that the most important contribution to diagnosis in the recognition of a segmental lesion is that it directs attention to the condition of the related bronchus; although it may prove to be normal, investigation of this matter can never be omitted when a segmental lesion persists or recurs, for then the bronchus is very likely to be abnormal, constricted or the seat of a newgrowth. One of the best examples of the value of segmental anatomy is to be seen in association with bronchial carcinoma. Here, when the X rays show a large pulmonary opacity, it may be thought that the entire shadow is due to a growth; but the growth itself is often small and the rest of the shadow due to consolidation or deflation of the pulmonary segment supplied by the affected bronchus. Thus recognition of the segmental nature of the lesion not only ensures prompt diagnosis and often indicates operability, but also it enables irradiation to be concentrated on the small area where the growth is situated. Another important application of segmental anatomy, according to Foster-Carter and Hoyle, is in the diagnosis, localization and external drainage of a pulmonary abscess, when it is desirable to recognize in which segment of the lung the abscess is situated, so that normal lung tissue is not transgressed during the operation. In pulmonary tuberculosis, shadows of segmental distribution in the skiagrams of adults often indicate an affection of a bronchus. L. G. Blair is quoted by Foster-Carter and Hoyle as stating that the truly segmental distribution of a pulmonary lesion is evidence against that lesion being a simple pneumonic process.

It is a corollary of the newer studies of broncho-pulmonary anatomy that in the future the skiagram of the lateral projection of the chest, in the past so little valued, will often assume importance equal to that of the postero-anterior projection in the radiological diagnosis of pulmonary disease. In fact it may well be that the present custom of making not a single skiagram of the patient's chest, but a pair of skiagrams for stereoscopic examination, is overdue for supersession. It is safe to say that in nine hundred and ninety cases out of a thousand a stereoscopic examination is never made after the skiagrams have left the radiologist, while in public hospital practice, as a rule only one skiagram is made. We suggest that a skiagram of the relevant lateral projection of the chest would be useful in diagnosis oftener than would a second skiagram of the postero-anterior projection.

FAT EMBOLISM.

FAT EMBOLISM is one of the rare hazards of trauma. War has provided an unusual experience for surgeons in many directions, and articles are appearing on this unusual condition as the result. Commander S. Warren has published a study of the records of 100 cases of fat embolism filed in the Army Institute of Pathology.[1] These cases occurred over a period of about four years, that is, from the period of general mobilization to the latter part of 1944. Ninety-one cases were due to trauma with fracture of one or more bones, four to blast injury without fracture, three to burns and one to pressure asphyxia without fracture. One might expect that the risk of embolism would be greater in proportion to the amount of marrow in the traumatized area, and this is so, for in 82% of cases following fracture involvement of the femur or tibia or both was present. Transport accidents have been a fruitful source of serious injury in this way; 61 of these cases were associated with motor vehicles. In practically all young healthy males were involved. Certain factors, such as obesity and hypertension, were not found to be significant in this survey, but one would not expect them to be. The important causal factors in producing fat embolism are the mobilization of fat, disruption and patency of veins and local pressure. All these are present in the injury of large bones when mobilization of a fractured limb occurs after local swelling takes place. It is harder to understand the occurrence of fat embolism after burns, but presumably the source of the fat is the damaged fat cells in the panniculus; pressure is possible from local œdema under thickened charred tissue or from pressure dressings. Warren agrees with other writers that the mere presence of even large amounts of fat in the blood stream will not produce embolism, and cites in support the non-occurrence of the condition in association with the high degrees of lipæmia found in diabetes. Apparently there is no danger of emboli being formed by the coalescence of very finely divided microscopic particles of fat in the blood. In fat embolism the level of the total blood fat is not necessarily high; in a few cases in this series some determinations were made of the blood fat, but no abnormal levels were found. But Warren points out that the fat contents of both arterial and venous blood are increased even a few minutes after trauma. It would appear that, quite apart from any arguments about the amount of fat needed to produce risk of embolism, the fracture of a single bone may set free enough liquid fat to cause this accident.

The previously published series have shown that fat embolism is a very rare accident, but the figures compiled from bombing casualties in this war point to a not inconsiderable frequency, at least from the standpoint of pathological diagnosis. Robb Smith is quoted as finding 41 instances of gross pulmonary fat emboli in 125 cases in which death followed injuries received during bombing raids. In 29 of the 41 cases pulmonary embolism was a major cause of death, but, of course, there are other important causes of embolism in the lungs than fat. Wakeley has reported finding fat embolism in varying degree in 40% of fatal cases of burns. The assessing of the importance of an excess of oily droplets in the pulmonary circulation at autopsy is not easy. In acute cases œdema of the lungs will also be found, and perhaps petechial hæmorrhages in the brain, and other evidences of asphyxia. Microscopic examination should show a wide distribution of fat droplets in the capillary system, particularly of the lungs and kidneys. There should be little doubt when such findings are clearly defined, and the diagnosis may be established even when the fatal issue has been delayed for many days. Impaired renal function does not seem to be significant in causing death. Clinically the occurrence of pulmonary or cerebral symptoms after fractures or extensive injury to soft parts, or after manipulation of a fractured limb, may suggest fat embolism. Fat may later be demonstrated in the urine or the sputum in some cases. Warren in recounting these features remarks on the free period after trauma during which no definite symptoms are apparent, though the patient may show apprehension. This latent period was noted specially by H. J. Nightingale, who reported a case last year.[1] He quoted the aphorism that alarming symptoms occurring at once or after a few hours are due to shock, after a few days to fat embolism, and after a week or weeks to the ordinary form of pulmonary embolism. He laid stress on the diagnostic importance of sudden collapse with cyanosis and pin-point pupils and perhaps later hyperpyrexia. Petechiæ on the skin should be looked for. There is little to be done once the accident occurs, but the avoidance of undue manipulation and the prevention of anoxia are measures that have a prophylactic value.

[1] *The American Journal of Pathology*, January, 1946.

PROFESSOR G. R. CAMERON IS HONOURED.

AMONG those recently recommended by the Council to be Fellows of the Royal Society is Professor G. R. Cameron, a graduate of the University of Melbourne, who was for a time assistant director of the Walter and Eliza Hall Institute, Melbourne, and is now professor of morbid anatomy at University College Hospital Medical School, London. His special wartime investigations into the effect of tannic acid on the liver and the toxicity of DDT were of particular value. All who know Professor Cameron and his work will acclaim this honour. We offer him warm congratulations.

[1] *British Medical Journal*, October 20, 1945.

Abstracts from Medical Literature.

PÆDIATRICS.

Penicillin in Infections of Infancy.

EDWARD C. ROSS COUPER (*Archives of Disease in Childhood*, September, 1945) draws attention to the high incidence of parenteral infection in infancy, however its role in the production of morbidity and mortality rates is interpreted. Many statistics are available in the literature. One of the latest surveys, for example, is that by Alexander and Elser (1944), who found evidence of parenteral infection in 88·6% of their cases, the bulk of this infection taking the form of upper respiratory infection and *otitis media*. The common organisms were hæmolytic streptococci and *Staphylococcus aureus*. Parenteral infection is even more deadly in the premature infant whose powers of resistance are feeble. In Walton Hospital, Liverpool, evidence of parenteral infection has been found in one-third of premature infants coming to post-mortem examination. When penicillin became available for civilian use, it was thought that parenteral infection in infancy might be a good field for its use, and immediate steps were taken to test this possibility. Twenty cases are described. The author states that this is a small number, but as this is a preliminary report, he thinks that perhaps the novelty of the treatment will excuse this defect. Owing to the necessity for economy in the use of penicillin, the patients have been selected as those most likely to benefit, and they fall into two groups. The first group is that of premature infants. These include: (*a*) The premature infant which goes steadily downhill. This type is frequently found at post-mortem examination to have some parenteral infection. (*b*) The premature infant which begins to have cyanotic attacks about ten days after birth, often a manifestation of bronchopneumonia. The second group comprises mature infants. In this group the parenteral infections which occurred happened to be all cases of *otitis media* manifesting itself by diarrhœa, vomiting, loss of weight, varying degrees of dehydration and signs of inflammation in the tympanic membranes. The penicillin was administered systemically, and 25,000 units were given daily, both to premature and mature infants. This amount of penicillin was made up in bottles containing 8·0 millilitres of saline solution and was given in 1·0 millilitre doses every three hours by intramuscular injection into the buttocks, each bottle lasting for twenty-four hours. Dehydration was combated by the administration by mouth of glucose and saline solution (1/5 normal saline solution and 5% glucose). Of the seven infants in the first group, five were thought to suffer from bronchopneumonia, manifested in four instances by the occurrence of cyanotic attacks about ten days after birth. In four of these cases the administration of penicillin coincided with improvement and the results were most satisfactory. In the fifth case no response took place and numerous coliform bacilli were found in the affected lung at post-mortem examination. MacGregor (1939) finds that in neonatal pneumonia the two most common and most lethal organisms are the *Staphylococcus aureus* and organisms of the colon bacillus group. It is possible that this type of pneumonia is due at first to organisms sensitive to penicillin, such as the *Staphylococcus aureus*, but that in some cases this infection cannot be overcome and a terminal invasion by *Bacillus coli*, insensitive to penicillin, leads to a septicæmic condition. The remaining three patients were premature infants who were going steadily downhill. Here again improvement coincided with administration of penicillin, but the nature of the lesion from which they suffered is unknown. The amount of penicillin given depended on clinical improvement. In the second group (twelve cases) gain in weight did not always follow administration of penicillin, but there was evidence of resolution of the inflammatory process in the middle ear in the majority of the cases and general clinical improvement occurred. In nine of these cases of *otitis media* the response to penicillin was satisfactory and there was often an immediate gain in weight and improvement in the general condition. In Case VII the *otitis media* settled clinically in five days, but the general condition remained unsatisfactory and improved only after intravenous treatment. In Cases VIII and X relapse occurred. The first of the patients in these two instances responded to the second course of penicillin, but the second died, although the inflammation appeared to have settled in three days. In those who recovered, the *otitis media* settled clinically in three to sixteen days, an average of 6·5 days. The decision to continue or discontinue penicillin was made on the appearance of the tympanic membranes, which were inspected daily, and in two cases mistakes were made, that is, in the two cases in which relapse occurred (VIII and X). Satisfactory gain in weight did not follow the first course of penicillin in these instances, which suggests that the inflammation had not completely subsided and that penicillin should have been continued for a longer period. The appearance of infantile tympanic membranes is often deceptive and such errors are unavoidable. The total dose varied between 100,000 units and 350,000 units. Six patients were dehydrated in varying degree, but responded well to the treatment described, only one requiring intravenous treatment. In the other instances, breast feeding was continued during penicillin therapy, one of the advantages of this form of treatment over the sulphonamides. The most striking features of this experiment are as follows: (i) No local treatment was given or was necessary. (ii) No spontaneous perforation of the tympanic membranes occurred. (iii) No myringotomies or mastoidectomies became necessary. The author compares this series with a previous series of twenty patients suffering from *otitis media* treated with sulphadiazine. In only four of these did the inflammation subside with the drug alone, two drums ruptured spontaneously after the commencement of the drug therapy, and four required myringotomy and two mastoidectomy. The two groups were unfortunately not concurrent and possibly there may have been some epidemiological difference between them. Nevertheless, the results in the penicillin group seem better than those in the sulphadiazine group. In the case in which death occurred the post-mortem findings were not satisfactory, but it is possible that an invasion by *Bacillus coli*, such as can occur in the first month of life, caused a septicæmia which was responsible for death. The encouraging results described suggest that penicillin will be useful in parenteral infection in infancy, but much further investigation is necessary to establish its exact scope.

Penicillin Treatment of Pneumococcus Meningitis in Infants.

GRETCHEN HUTCHINS AND JOHN A. V. DAVIES (*Journal of Pediatrics*, December, 1945) have analysed the results of the penicillin treatment of fourteen cases of pneumococcus meningitis occurring in children under two years of age between September 20, 1943, and December 28, 1944. The employment of penicillin as a new agent in the treatment of pneumococcus meningitis calls for a revaluation of the prognosis and a discussion of the therapeutic problems involved. Previous reports on the use of penicillin in this formidable disease have dealt chiefly with adults or older children. Seven of the fourteen patients had had some form of sulphonamide at various intervals before their admission to hospital. The indications were usually described as sore throat or pneumonia, but in one case pneumococcus meningitis was known to be present. In at least four of them the dosage schedule was too low for the treatment of a severe infection. The therapy in this hospital, while fundamentally similar in all these cases, was varied from time to time for special reasons. Three of the patients appeared so ill at the time of admission that they were given intravenously single doses of sodium sulphadiazine as soon as the lumbar puncture had been made and blood culture attempted. In six other cases sulphadiazine was given parenterally or orally at various times throughout the course of the illness for complications or for other reasons associated with the meningitis. If sulphadiazine was used an attempt was made to keep the blood levels about 12 and preferably between 15 and 20 milligrammes *per centum* in early therapy. Penicillin was given intramuscularly on a dosage schedule of 3,000 units every three hours in the earlier cases and to smaller infants. The other patients were given 5,000 units every three hours. The schedule of intrathecal therapy was 10,000 units twice a day (or three times a day for the first few days) in an aqueous or saline solution containing 1,000 units per millilitre. In the earlier cases the dosage was somewhat less, 2,500 to 5,000 units two or three times a day. The dosage sometimes departed from this schedule because of mechanical difficulties encountered which limited the amount of spinal fluid removed. When blocks occurred so that spinal fluid could not be obtained by the lumbar route, penicillin was given intracisternally when possible, and, in a few cases, into the ventricle. Penicillin was administered by the intrathecal route for an average of eleven days, but the extremes were from one to sixteen days. In some cases it was given intermittently because of the mechanical

difficulties already mentioned, and in one because cultures were again obtained from the spinal fluid. The patient in the last-mentioned case died with a complicating infection. In four of the cases an attempt was made to replace most of the spinal fluid with oxygen in the hope of lessening the formation of subarachnoid adhesions. The data are insufficient for an evaluation of this procedure. Type-specific antipneumococcus rabbit serum was given in four of the cases. The indications were: uncertainty concerning the effectiveness of penicillin therapy in the first case, the severity of the infection in the second, and the youth of the patients and previous disappointing results with penicillin in the remaining two. The authors conclude that this series of infants illustrates the favourable results which the addition of penicillin to the therapy of pneumococcus meningitis has achieved. Recoveries, either intact or with residual damage to the brain, were significantly more frequent than with sulphonamides and type-specific antiserum. Of fourteen infants, nine survived. However, five of these nine exhibited varying degrees of permanent central nervous system damage. The younger the infant and the longer the duration of the infection, the poorer the prognosis. (Four of the fourteen patients whose cases are presented were under two months of age; two of the deaths were of older infants ill two weeks before therapy.) Adequate penicillin therapy can sterilize the cerebro-spinal fluid and blood stream within twenty-four hours if it is administered before heavy fibrinous exudate is formed in the meninges. Penicillin therapy should probably be continued about one week after sterilization of the spinal fluid, and sulphonamide therapy for a week or more longer if extrameningeal foci of infection are present or suspected. In the presence of heavily infected spinal fluid, spinal drainage and replacement therapy with penicillin and oxygen, and possibly other special procedures may be advisable. These measures await further clinical trial. The value of type-specific antiserum, in conjunction with penicillin and an appropriate sulphonamide, such a sulphadiazine, has not as yet been sufficiently appraised.

ORTHOPÆDIC SURGERY.

Chronic Dislocation of the Base of the Metacarpal of the Thumb.

S. W. N. EGGERS (*The Journal of Bone and Joint Surgery*, July, 1945) describes his operation for correction of chronic dislocation of the base of the metacarpal bone of the thumb. An incision is made on the dorsum of the hand, extending from a point proximal to the articulation of the first metacarpal and the greater multangular distally between the first and second metacarpals. Care is taken to preserve the digital sensory nerves. The radial artery is ligated where it perforates the intermetacarpal space. The first metacarpal is liberated at the base and the fibrous tissue is removed. The author states that the dislocation can then be reduced, but that stability and retention in the reduced position are not easy to maintain. He utilizes a portion of the tendon of the *extensor carpi radialis longus* as a ligament to maintain reduction. This tendon, which is inserted into the base of the metacarpal of the index finger, is in two portions—medial and lateral. The tendon is drawn down from its sheath and the lateral portion is divided sufficiently to secure a band of adequate length. A hole is drilled through the base of the metacarpal of the thumb close to the articular surface. The band of tendon is then passed through the drill hole and sutured under tension to itself with silk. A plaster cast is applied for three weeks. The author states that the return of function is satisfactory.

Plasmocytoma of Bone.

WILLIAM TENNENT (*The British Journal of Surgery*, April, 1945) presents evidence to the effect that plasmocytoma of bone forms a distinct clinical picture of a solitary intramedullary neoplasm which remains strictly localized for several years, but if untreated develops into the fatal condition of multiple myelomatosis. The fifty cases recorded in the literature are analysed. Of the affected persons, 70% were men. The average age at the time the diagnosis was established was fifty-one years. The distribution of affected sites was as follows: pelvis, 16 cases; femur, 12; humerus, 9; vertebræ, 6; skull, 5; tibia, 1; clavicle, 1. The usual symptoms were pain, local swelling and pathological fracture. The prognosis is less favourable than in osteoclastoma and considerably more favourable than in osteogenic sarcoma. In the case reported by the author the growth on the ilium, without receiving any treatment whatever, apparently advanced slowly and remained localized for four and a half years before the onset of multiple myelomatosis. The author raises the question whether multiple myelomatosis is a systemic disease of the bone marrow with multicentric origins, or whether it merely represents the terminal stage of multiple metastasis from a primary plasmocytoma.

The Kuntscher Method of Treatment of Fractures.

B. M. TORDOIR AND E. J. MOEYS (*The Journal of the American Medical Association*, July 14, 1945) point out that the best type of fracture for the Kuntscher treatment with a steel nail is a transverse fracture in the diaphysis. The fracture is accurately reduced with the help of a special frame under fluoroscopic control. For the femur an opening is made in the trochanteric fossa just medial to the greater trochanter with a piecer, and a thin stiff wire is pushed toward the medullary cavity beyond the fracture and into the distal fragment. The Kuntscher nail is then hammered over the wire and the wire is removed. A few days after the operation, which is performed as soon as possible after the accident, the patient is able to walk about and to leave hospital. When consolidation of the fracture occurs the perforated head of the nail which remains outside the bone is "hooked" with a special instrument, and without any force the nail is easily removed. The authors have used this method for 34 patients with closed fractures of femur, tibia, humerus, radius and ulna, fibula and olecranon. They have also used this method for ten patients with compound fractures, and in no case has osteomyelitis occurred. The authors state that since Kuntscher introduced this method in 1940 he has treated many patients with satisfactory results, and in 1944 Böhler in a third edition to his book recorded the results in 500 cases.

The Treatment of Non-Union or Delayed Union of Fractures of Long Bones.

D. M. MEEKISON (*The Journal of Bone and Joint Surgery*, July, 1945) discusses the operative technique employed in the treatment of delayed union or non-union of fracture of long bones. The author uses a tourniquet wherever possible, even in grafting fractures of the femoral shaft. In fractures of the shaft of the femur a Steinmann pin is passed from front to back just above the greater trochanter and just inside the iliotibial band. The tourniquet is then applied around the groin and above the pin. The author believes that all fibrous tissue between the fragments and all excess callus should be removed. The ends of the fragments are freshened by the removal of sclerotic bone and the medullary canals are opened by drilling. When the fragments have been accurately opposed, a bed is prepared by a chisel. The bed is prepared on the most readily available surface, except in the case of the tibia where the bed is made over the lateral aspect of the bone, in which site the graft will be covered by the extensor muscle bellies. The author considers that it is preferable to obtain grafts from the upper end of the tibia, since these grafts seem to be more osteogenic. Furthermore the nutrient artery is avoided as well as the possible subsequent development of a hæmatoma. In addition the removal of a graft from the upper end of the the tibia provides easy access to cancellous material. When the graft has been removed from the donor tibia the medullary portion is removed from the cortical portion by means of a broad osteotome. The medullary portion of the graft is placed in the medullary canal and the cortical portion is settled in the prepared bed. The graft is fixed in position by vitallium screws which must engage both cortices of the recipient fragments. The author uses six screws in all cases except forearm grafts where four screws are employed. The screws are "staggered" in order to ensure against torsion strain. All crevices are then filled with cancellous material (scooped from the donor tibia), the material being used "like mortar". A padded plaster cast is applied. After fourteen days this cast and the sutures are removed and a non-padded cast is substituted. At the same time the sutures are removed from the donor leg and a long non-padded cast is applied. The author considers that protection for ten weeks should suffice for the donor leg. The grafted limb is fixed in plaster until there is clinical and X-ray evidence of solid union. In the series of patients treated by the author with onlay grafts the average period of fixation was ten weeks for fracture of the radius, ten weeks for the ulna, four months for the femur, four months for the tibia and eleven weeks for the humerus. This method of treatment was used in the treatment of 170 patients with only one failure. This occurred with a patient who had an old ununited fracture of the upper end of the tibia; successful union occurred after a second similar graft had been inserted.

British Medical Association News.

THE OPHTHALMOLOGICAL SOCIETY OF AUSTRALIA (BRITISH MEDICAL ASSOCIATION).

ANNUAL MEETING.

THE fifth annual general and scientific meeting of the Ophthalmological Society of Australia (British Medical Association) was held at the Stawell Hall, Royal Australasian College of Physicians, Macquarie Street, Sydney, on October 3, 4 and 5, 1945, DR. N. MCALISTER GREGG, the President, in the chair. Those present included three members from Queensland, twenty-five members from New South Wales, seven members from Victoria and one member from South Australia.

President's Address.

The President, Dr. N. McAlister Gregg, in expressing relief and thankfulness at the ending of the war, pointed out that peace would bring forth difficult problems which would require for their solution a similar spirit to that which had actuated the United Nations in the bitter years. He congratulated the members on the preservation of the vitality of the society during the war period, and looked forward to a future of useful accomplishment. He said that the society would remember with pride and gratitude the work of members in the forces. It was the society's duty to make it possible for service medical officers to obtain in Australia all the specialist training in ophthalmology which they might need. Dr. Gregg stressed the importance of a high standard of teaching. Dr. Gregg continued with references to the part which members would take in the matter of industrial ophthalmology in its various aspects, and their obligations in research. In conclusion, he said that their motto might well be: "Raise your hat to the past, take off your coat to the future."

Vascular Diseases and their Relationship to the Eye.

DR. C. G. MCDONALD (Sydney) read by invitation a paper which dealt comprehensively with a variety of vascular diseases and their relationship to the eye. He discussed particularly diagnosis and prognosis and made a plea for a rational classification based on a generally accepted nomenclature. He pointed out that the various terminologies caused confusion in a discussion on vascular diseases and made a plea for the abandonment of the term "arteriosclerosis".

Iris Prolapse.

DR. N. M. MACINDOE (Sydney) described a technique for dealing with iris prolapse, involving the performance of a peripheral iridotomy. He considered that this method superseded the use of the cautery.

Toxoplasmic Encephalomyelitis and Chorioretinitis.

DR. E. GRAEME ROBERTSON (Melbourne) read by invitation a paper written by himself and DR. J. BRUCE HAMILTON (Hobart) on the subject of toxoplasmic encephalomyelitis and chorioretinitis, and illustrated by the cinematograph and lantern slides. Dr. Robertson gave an historical review of the relatively few papers which had established the nature of the causal organism (a protozoon toxoplasma). The clinical and pathological pictures were then described as a prelude to the description of a number of cases in which healing had occurred, in all with reduction of higher cerebral functions, epilepsy, microphthalmos, chorioiditis and intracerebral calcification. When the infection commenced in early life the nervous system and the eye bore the brunt of the attack; when it commenced in later life the manifestations were generalized and other organs were predominantly affected. The initiation of infection in the early months of gestation led to defective development of the eyes. Interesting comparisons with other infections of the fœtus were made.

Unilateral Pulsating Exophthalmos due to a Developmental Defect of the Orbital Wall.

DR. E. GRAEME ROBERTSON (Melbourne) read a further paper on the subject of unilateral pulsating exophthalmos due to a developmental defect of the orbital wall. This paper was also illustrated by the cinematograph. Dr. Robertson said that the condition was rare and little known, but it was characteristic, and its recognition would prevent misguided attempts at treatment. In a number of such cases exploration had been carried out in the belief that the cause was neoplastic. The radiological findings in one case were described; and on the basis of these an initial diagnosis of a vascular type of neoplasm had been made. However, encephalography showed that the subarachnoid space extended into the orbit, and suggested the presence of a developmental defect. Lumbar puncture findings, which were probably characteristic of the disease, were described. Tomographic radiographs showed the contrast between the two sides of the skull. Finally, confirmation of the diagnosis resulted from a search of the literature. There were certain interesting associations, notably with neurofibromatosis and hydrophthalmos.

Septic Thrombosis of the Cavernous Sinus Treated with Penicillin.

DR. R. G. BANKS-SMITH (Tamworth, New South Wales) gave a report of a patient affected by septic thrombosis of the cavernous sinus and treated with penicillin.

Contact Lenses.

DR. DARCY WILLIAMS (Sydney) read a paper giving a brief historical summary of contact lenses followed by a discussion on Zeiss type lenses. He then referred to individually moulded lenses and described a method of taking impressions of the eyeball with atomized wax. The use of acrylic resin for making lenses was discussed. Observations on the configuration of the eyeball led to a dissertation on the principles of the fitting of lenses. Disabilities experienced by wearers and the use of filling solutions were also mentioned.

The Treatment of Secondary Cataract.

DR. E. TEMPLE SMITH (Sydney) said that his paper was concerned with cataract as a sequel of chronic iritis. Two types were described. In one, all tendency to iritis was obsolescent. In the other, although no active iritis was present, there was some vascularity of the iris and the eye readily flushed on examination. The surgical and medical treatment of both types was discussed.

Premedication and Cooperation in Cataract Operation.

DR. F. GREGORY ROBERTS (Sydney) described his method of premedication in cataract operation and advanced arguments for its support. He averred that cooperation of the patient in cataract operation was not necessary. With adequate premedication the patient could sleep through the operation. A summary of dosages and drugs followed.

An Ocular Syndrome.

DR. R. G. WADDY (Sydney), in his paper, submitted that there was a definite syndrome characterized by poor distant visual acuity, hypermetropic astigmatism, nystagmus, an albinoid type of fundus, and a high degree of intellect.

Sarcoidosis.

DR. W. D. COUNSELL (Melbourne) read a paper prepared by himself and DR. J. BRUCE HAMILTON (Hobart) on the sarcoidosis of Boeck. Dr. Counsell reported two cases. In one case cure had been effected seven years earlier and in the other the condition was resolving. He said that the resulting chorioido-retinitis in one case could easily be confused with syphilitic chorioido-retinitis. The Mantoux test in both cases produced a negative result. The X-ray appearances of the lungs in one case resembled those of miliary tuberculosis, and after examination of lymph nodes taken in both cases a diagnosis of sarcoidosis was made.

DR. HUGH RYAN (Melbourne) presented a paper written by himself and DR. W. D. COUNSELL (Melbourne) continuing the discussion on ocular sarcoidosis. Dr. Ryan stressed the occurrence of ocular manifestations of Boeck's sarcoid, and made a short comment on the ætiology and pathology of the condition. Dr. Ryan said that uveoparotitis had a place amongst the diverse manifestations of sarcoidosis, and described a case in which features of both uveoparotitis and Boeck's sarcoid were present.

Traumatic Monocular Chronic Glaucoma.

DR. A. W. D'OMBRAIN (Newcastle) suggested that traumatic monocular chronic glaucoma was an entity which might be missed. No references to it had been found in any textbooks. If it was a clinical entity, it was one of importance both medically and legally—medically, because, in contradistinction to primary chronic glaucoma, the other eye would not become affected, and legally, because both soldiers

and civilians were at present refused compensation for chronic glaucoma despite a history of trauma. It was further contended that the condition was a true progressive glaucoma and not merely a static ocular hypertension, and that it might go on to a condition of acute congestive glaucoma if not controlled by treatment or operation. Dr. D'Ombrain put forward his argument for the existence of this condition on the evidence of eighteen cases from various sources.

Vitamin C (Ascorbic Acid) in Keratitis.

DR. G. H. BARHAM BLACK (Adelaide) referred to some reports in recent medical literature of the effect of vitamins on ocular physiology and pathology, and gave an account of three cases of corneal ulceration occurring in patients who were found by laboratory tests to be grossly deficient in vitamin *C*. Dr. Barham Black laid emphasis on the nature of the corneal lesions, superficial ulcers extending from the limbal zone centrally and followed by healing and vascularization in the same direction, and he likened them to a Mooren's ulcer. Treatment with ascorbic acid therapy was followed by rapid healing.

Annual Dinner.

The annual dinner, held at the Hampton Court Hotel, Darlinghurst, was well attended and proved a most enjoyable function.

Annual General Meeting.

Apart from general business of the society, the chief matter discussed at the annual general meeting was post-graduate education of ex-servicemen in ophthalmology.

Dr. J. Ringland Anderson (Victoria) was elected president for the ensuing year

It is proposed to hold the sixth annual general and scientific meeting at Melbourne from October 20 to 26, 1946.

THE KATHERINE BISHOP HARMAN PRIZE.

THE Council of the British Medical Association is prepared to consider an award in 1947 of the Katherine Bishop Harman Prize. The value of the prize is £75.

The purpose of the prize, founded in 1926, is the encouragement of study and research directed to the diminution and avoidance of the risks to health and life that are apt to arise in pregnancy and child-bearing. It will be awarded for the best essay submitted in open competition, competitors being left free to select the work they wish to present, provided this falls within the scope of the prize. Any medical practitioner registered in the British Empire is eligible to compete.

Should the Council of the Association decide that no essay submitted is of sufficient merit, the prize will not be awarded in 1947, but will be offered again in the year next following this decision, and in this event the money value of the prize on the occasion in question shall be such proportion of the accumulated income as the Council shall determine. The decision of the Council will be final.

Each essay must be typewritten or printed in the English language, must be distinguished by a motto, and must be accompanied by a sealed envelope marked with the same motto and enclosing the candidate's name and address. Essays must be forwarded so as to reach the Secretary, British Medical Association House, Tavistock Square, London, W.C.1, not later than December 31, 1946. Inquiries relative to the prize should be addressed to the Secretary.

NOTICE.

THE General Secretary of the Federal Council of the British Medical Association in Australia has announced that the following medical practitioners have been released from full-time duty with His Majesty's Forces and have resumed civil practice as from the dates mentioned:

Dr. H. N. Merrington, 49, Belmore Road, Randwick (March 1, 1946).
Dr. F. H. M. Callow, 143, Macquarie Street, Sydney (April 24, 1946).
Dr. J. V. Champain, Molesworth Street, Tenterfield, New South Wales (March 4, 1946).
Dr. L. S. Loewenthal, 217, Macquarie Street, Sydney (May 1, 1946).

Medical Societies.

MELBOURNE PÆDIATRIC SOCIETY.

A MEETING of the Melbourne Pædiatric Society was held on August 8, 1945, at the Children's Hospital, Melbourne, DR. H. DOUGLAS STEPHENS, the Acting President, in the chair. Part of this report appeared in the issues of January 26 and February 2, 1946.

Cœliac Syndrome.

DR. GEORGE SCOTT read a paper entitled "Cœliac Syndrome: A Survey from the Children's Hospital, Melbourne" (see page 659).

DR. J. W. GRIEVE said that the society was grateful to Dr. Scott for the pains to which he had gone to prepare his paper. When Dr. Grieve twelve months earlier had made the suggestion that this work be instituted, he was anxious to learn just what happened to these children. In the past, it had been an easy matter to diagnose the true "cœliacs"; but many other cases were met with, especially among smaller babies, who did not quite fit into the picture, though presenting some of the features of the disease. A quicker recovery and a greater susceptibility to upper respiratory tract infections were two of the differences. Dr. Grieve said that he was grateful to Dr. Scott for elaborating the subject and attempting to classify the cases in some order, to enable the problem to be attacked more intelligently.

DR. KATE CAMPBELL thanked Dr. Scott for his interesting paper and for the great list of references. She said that she was interested in the role of the adrenal gland in the absorption and synthesis of fat, and would be grateful for a list of references pertaining to that subject.

Naval, Military and Air Force.

APPOINTMENTS.

THE undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Numbers 58, 64 and 69, of March 28, April 4 and 11, 1946.

PERMANENT NAVAL FORCES OF THE COMMONWEALTH (SEA-GOING FORCES).

His Royal Highness the Governor-General in Council has approved of the following appointment being terminated: Acting Temporary Surgeon Lieutenant-Commander (D) John McFarlane Wark, 1st February, 1946.

Thomas Bowen Ready is appointed Surgeon Lieutenant (for short service), dated 25th February, 1946.

AUSTRALIAN MILITARY FORCES.

Australian Army Medical Corps.

VX132560 Lieutenant-Colonel R. G. Worcester, E.D., relinquishes command of 2nd/1st Australian Casualty Clearing Station and is appointed to command 2nd/9th Australian General Hospital, 4th February, 1946.

SX25140 Lieutenant-Colonel E. F. Gartrell is placed upon the Regimental Supernumerary List, 4th February, 1946.

SX27204 Major W. R. C. Morris is placed upon the Regimental Supernumerary List, 15th February, 1946.

NX146280 Captain J. F. McInerney is removed from the Regimental Supernumerary List, 31st January, 1946.

The following officers are transferred to the Reserve of Officers (Australian Army Medical Corps): Lieutenant-Colonels SX14109 A. C. McEachern, 4th January, 1946, NX217 A. R. Colwell, 10th January, 1946, WX3416 G. C. Moss, 12th January, 1946, QX40894 P. W. Hopkins, M.C., 18th January, 1946, SX21585 T. A. B. Jones, 24th January, 1946.

To be Lieutenant-Colonels, 27th September, 1945.—Majors (Temporary Lieutenant-Colonels) VX48607 T. C. Backhouse, NX217 A. R. Colwell and WX3416 G. C. Moss.

102nd Australian Convalescent Depot.—QX22828 Major R. S. Bennett is placed upon the Regimental Supernumerary List, 13th December, 1945.

Inter-Service Medical Wing Demobilization Centres (Australian Military Forces Component).—VX104252 Captain (Temporary Major) D. R. McFarlane is placed upon the Regimental Supernumerary List, 23rd December, 1945.

The appointments of the following officers are terminated on demobilization with effect from the dates indicated:

Temporary Wing Commander J. C. Laver (251199), 17th October, 1945, Temporary Flight Lieutenant B. Starr (257522), 29th October, 1945.

Reserve of Officers.

The undermentioned officers are transferred to the Reserve of Officers with effect from the dates indicated, and, where applicable, cease to be seconded. Officers holding temporary rank relinquish such temporary rank on the date of transfer to the Reserve of Officers:

WX3355 Major B. St. P. Gillett, 11th January, 1946, WX17088 Major L. W. Martin, 10th January, 1946, and Captains N274186 G. L. Gulson, 4th December, 1945, NX71041 J. Gribben, 28th November, 1945, and WX23056 A. A. W. Roe, 18th January, 1946.

2nd/1st Australian General Hospital.—QX19165 Captain C. W. Taylor, 4th January, 1946.

2nd/5th Australian General Hospital.—NX443 Major J. Kingsley, 1st January, 1946, QX40885 Major J. R. Adam, 4th January, 1946, and NX144608 Captain J. L. Dowling, 2nd December, 1945.

No. 102 (Holland Park) Military Hospital.—QX59539 Captain R. S. Cohen, 8th January, 1946, and Q108314 Captain W. Monz, 20th December, 1945.

103rd Australian General Hospital.—Majors NX87 P. A. Tomlinson, O.B.E., 21st November, 1945, and NX34757 H. B. Woolford, 11th December, 1945.

No. 110 (Perth) Military Hospital.—WX34808 Major O. R. Corr, 18th January, 1946.

No. 112 (Brisbane) Military Hospital.—Majors QX35632 I. H. MacGillivray, 8th January, 1946, and NX12278 W. S. L. Stening, 21st November, 1945.

No. 113 (Concord) Military Hospital.—Majors NX450 W. D. Sturrock, 23rd November, 1945, and NX70335 R. H. B. Bettington, 5th December, 1945.

No. 114 (Goulburn) Military Hospital.—NX34921 Major C. H. W. Lawes, 22nd November, 1945.

118th Australian General Hospital (Australian Imperial Force).—NX243 Major E. L. Davis, 24th November, 1945.

22nd Australian Camp Hospital.—NX103438 Major C. W. England, 23rd November, 1945.

67th Australian Camp Hospital.—Q119524 Captain L. T. Moran, 9th January, 1946.

2nd Australian Women's Hospital.—NX153001 Major A. M. B. Grant, 13th January, 1946.

2nd/4th Australian Field Ambulance.—NX76354 Major L. Rail, 4th December, 1945.

19th Australian Field Ambulance (Australian Imperial Force).—Captains NX115904 C. H. Wood, 15th January, 1946, and NX118230 C. H. Thomson, 6th January, 1946.

2nd Australian Sea Ambulance Medical Staff (Australian Imperial Force).—NX118813 Captain P. L. Charlton, 11th December, 1945.

Inter-Service Medical Wing Demobilization Centres (Australian Military Forces Component).—Majors QX40814 G. R. Kurrle, 8th January, 1946, and VX15914 C. D. Donald, 4th December, 1945.

NX70579 Major F. P. C. Claffy, 23rd November, 1945, Captains NX34756 N. H. Rose, 1st January, 1946, NX77245 A. F. Smith, 28th November, 1945, VX39059 R. W. Greville, 30th November, 1945, and WX11057 T. Godlee, 16th January, 1946.

6th Australian Camp Hospital.—NX119097 Captain (Temporary Major) K. Klein, 13th December, 1945.

ROYAL AUSTRALIAN AIR FORCE.

Permanent Air Force: Medical Branch.

The appointment of Temporary Wing Commander, Acting Group Captain C. J. N. Le Leu, O.B.E. (1176), is terminated on demobilization with effect from 4th December, 1945.

Citizen Air Force: Medical Branch.

Group Captain G. C. Willcocks, O.B.E., M.C., M.B., Ch.M. (Sydney), F.R.C.P. (London), M.R.C.P., F.R.A.C.P., is appointed Honorary Physician to His Royal Highness the Governor-General, with effect from 1st March, 1946, for a period of three years.

Wing Commander C. H. C. Searby, B.Sc., M.B., M.S., F.R.C.S., F.R.A.C.S., is appointed Honorary Surgeon to His Royal Highness the Governor-General with effect from 1st March, 1946, for a period of three years.—(Ex. Min. No. 94—Approved 3rd April, 1946.)

The appointments of the following officers are terminated on demobilization with effect from the dates indicated: Temporary Squadron Leader H. M. Webber (261893), 18th October, 1945, Temporary Flight Lieutenant, Acting Squadron Leader T. E. H. Spark (267437), 7th November, 1945, Temporary Flight Lieutenant D. Gilmour (423707), 29th November, 1945.

The appointments of the following Temporary Squadron Leaders are terminated on demobilization with effect from the dates indicated: R. G. Mackay (261769), 22nd October, 1945, B. W. Monahan (261612), 2nd November, 1945.

The appointments of the following officers are terminated on demobilization with effect from the dates indicated: Temporary Wing Commander F. F. Ellis (261200), 13th November, 1945, Temporary Squadron Leader, Acting Wing Commander G. S. Colvin (261246), 28th November, 1945. (Temporary Squadron Leaders) T. Burfitt (261204), M. A. O'Halloran (262870), 26th November, 1945, M. T. Drummond (264589), W. G. Galley (262375), 28th November, 1945, W. P. H. Dakin (261274), 4th December, 1945.

The appointment of Temporary Squadron Leader, Acting Wing Commander S. B. Forgan (281237) is terminated on demobilization with effect from 3rd January, 1946.

The appointments of the following officers are terminated on demobilization with effect from the dates indicated: Temporary Flight Lieutenant, Acting Squadron Leader E. D. E. E. O'Brien (257096), 1st December, 1945, Temporary Flight Lieutenant H. R. Hawkins (251777), 23rd November, 1945.

Reserve: Medical Branch.

Temporary Squadron Leader K. S. Harrison (261888) is transferred from the Active List with effect from 23rd October, 1945.—(Ex. Min. No. 85—Approved 3rd April, 1946.)

Temporary Squadron Leader R. W. Hazelton (261261) is transferred from the Active List with effect from 21st November, 1945.—(Ex. Min. No. 87—Approved 3rd April, 1946.)

Temporary Flight Lieutenant J. M. Barrett (254593) is transferred from the Active List with effect from 27th October, 1945.—(Ex. Min. No. 93—Approved 3rd April, 1946.)

Special Correspondence.

NEW ZEALAND LETTER.

By OUR SPECIAL CORRESPONDENT.

Addition to Benefits under Social Security—Laboratory Diagnostic Services.

From April 1, 1946, laboratory diagnostic services performed by hospital board laboratories and by private pathologists become the subject of benefits. Certain types of examination are specifically excluded, such as specimens for public health services, post-mortem examinations, laboratory service for dental purposes and for purposes of life insurance and the preparation of sera and vaccines.

The general laboratory work that is provided for is to be done without cost to the doctor or to the patient, but doctors must requisition the examinations in writing to make the claim valid.

The hospital and private pathologists are somewhat apprehensive of this measure. There is still a very great relative shortage of men and material in the world of pathology, and unless medical men are very circumspect in the requests they make, the present arrangements may become very overloaded and lead to lower standards of work, if not actual breakdown.

Correspondence.

YAWS AND BERIBERI: A WARNING.

SIR: May I, as a result of years of experience in the islands, make a couple of warnings that may be useful.

1. In a capital city tram recently I saw a young married woman suffering from a typical case of florid secondary yaws on the face. The lesions were smeared with a white ointment and had obviously been mistaken for impetigo, a very natural mistake where yaws is unknown. Yaws where endemic is universal and acquired by inoculation, purposeful or accidental, in small childhood. But in non-immune adults it is as much a venereal disease as syphilis, and has been so acquired by a few whites. Though so closely related to syphilis, it differs greatly in response to drugs. The best

cure still is a full course of arsenicals of the neoarsphenamine type. Check by means of one or other of the complement tests. The main thing to remember is that the arsenoxide type of antisyphilic arsenicals are not at all satisfactory in yaws, whatever they may be in syphilis.

2. Beriberi. The myocardial damage in this disease is much more extensive than seems to be realized. Any islands resident has known that natives with a history of beriberi had a way of dying of sudden heart failure following small illnesses and exertions, even though they appeared to have completely recovered from their beriberi long before. I think all ex-beriberi patients should be examined with the electrocardiograph several times and warned against sudden exertion for two or three years. Also the risk and need for extra rest and care in the case of febrile illness should be explained to them.

Yours, etc.,

C. M. DELAND, M.B., B.S., D.P.H., D.T.M.
302, Goodwood Road,
Clarence Park,
South Australia.

April 6, 1946.

A PSYCHIATRIC PROGRAMME FOR PEACE.

SIR: In his article "A Psychiatric Programme for Peace", Dr. Ellery discusses the mental malady which has come upon the world. He describes the ætiology as a disproportionate attention to the physical sciences at the expense of the humanities and the science of human conduct. As remedy he prescribes the application of knowledge concerning the hidden springs of human conduct.

Dr. Ellery is in good company. Socrates himself made "know thyself" the foundation of his philosophy for life. The new knowledge confirms older insights concerning human nature. But with the quest for and use of knowledge, man must also (as the Greek myths taught) renounce the "hybris" (pride) which refuses to accept the obligations and necessities which derive from man's creaturely condition, the pride which seeks to make himself a god. He must also accept another standard than the satisfaction of his desires as the final arbiter of conduct.

If he does not do so, the consequences of this new knowledge, as of every other knowledge not subjected to these obligations, are not problematic. They are already with and over us—a sword more dangerous and precarious than the sword of Damocles—the atomic bomb.

There is an old story invested at least with the authority of the wise myth. A woman was deceived by false and seductive assurances and attracted by manifest utilitarian and æsthetic values and by the even greater attraction of the active principle of sagacity. As the story puts it—"she saw the tree of the knowledge of good and evil that it was good for food, a delight to the eyes, and to be desired to make one wise". She thought to exploit these boons and yet escape the mortal consequences of self-will and disobedience.

Wisdom teaches that humanity's deeper problems are in the realm (to use the language of Dr. Ellery's specialty) not of cognition (knowledge) but of conation (purpose and desire). This also is the import of the well-known aphorism of the philosopher Kant: "There is nothing which is always and altogether good but a good will."

The Nazis were wise as serpents. They knew a devil of a lot. They proved themselves dynamic psychologists; but they brought chaos to the world and led Germany to hell. It has been widely taught that Germany, with her delusions of persecution and grandeur, presented the symptoms of a national paranoia. What was needed was not, fundamentally, more psychological knowledge. The Nazis were well informed about the dark atavistic trends in human nature. Germany needed (in the language of the Greek New Testament) a metanoia, that is, an after-thought, a different choice, a repentance.

Yours, etc.,

CHAS. I. MCLAREN.
4, Barry Street,
Kew, E.4,
Victoria.

April 15, 1946.

Post-Scriptum.

Today is Good Friday. The events of that day and their upshot demonstrate an energy in history which has effected such a "metanoia" in individuals and in societies. They provide assurance that, linked with the new knowledge and the power over nature which the physical sciences bestow, the goal Dr. Ellery seeks is attainable.—C.I.McL.

SPONDYLITIS ANKYLOPOIETICA.

SIR: It is gratifying to see that "Froach Eilean" is interested in this subject, and I hope that the interest is not occasionel altogether by his own unfortunate experience. In that I can sympathize, since, as he surmises rightly, I have had unwanted familiarity with the same complaint for nearly thirty years. In fact my first personal introduction to spa treatment was at the ripe age of ten.

As your correspondent states, some few cases of *spondylitis ankylopoietica* do not proceed to complete ankylosis, but I am somewhat doubtful about the exact diagnosis of these until I can accumulate a sufficiently large series. Some of them are of a "mixed" type. I still believe that in New South Wales at any rate *spondylitis ankylopoietica* is a rare disease by comparison with *spondylitis osteoarthritica*, and does not cause anything like as much continual suffering, although the ultimate disability may be more dramatic in appearance. Many practitioners confuse the two main forms of arthritis, and I have often encountered cases of hypertrophic arthritis diagnosed as "infective" or "atrophic" and *vice versa*.

Another, and very timely, point suggested by "Froach Eilean's" letter—the benign *spondylitis osteoarthritica*, *spondylitis osteoarthritica* is not benign—far from it. It is by far the commonest spinal complaint today, and the most persistently painful and morale-destroying. It can, in the long run, prove more detrimental to the patient's well-being and work capacity than *spondylitis ankylopoietica*. It is not particularly a disease of old age, as patients are often told. Radiological and post-mortem examinations in Germany showed the incidence of spinal osteophytes to be as follows:

11% of age group	.....	20 to 30 years
36% of age group	.....	30 to 40 years
78% of age group	.....	40 to 50 years
93% of age group	.....	over 50 years

(G. Schmorl: *"Archiv und Atlas der normalen und pathologischen Anatomie in typischen Röntgenbild"*, 1932.)

What might be called benign, in so far as they may be symptomless, is the presence of osteophytes on the vertebral margin (so-called marginal polyspondylitis). These cause no pressure symptoms, although they may eventually lock the vertebræ. But osteophytes of the intervertebral articulations (so-called hypertrophic spondylarthritis) can cause very constant pain. Most common of all is the ligamentitis and fibrositis which is both a precursor and concomitant of osteospondylitis, and which causes radicular pain and almost every other form of backache. In my opinion, fibrositis is to osteoarthritis what sacro-iliitis is to *spondylitis ankylopoietica* (according to Scott). It is the danger signal and the indication for preventive treatment.

One more point. Anatomy and pathology are no doubt the main pillars of the practice of medicine, but I feel that as a profession we are too pathologically minded and not sufficiently health-conscious. It is true that we deal with the morbid in life, and that, as Bernard Shaw remarked, our existence is dependent on that factor, but a little broader concept of the various factors that make for health is, I think, overdue. More especially is this so in treating the rheumatic diathesis.

Yours, etc.,

E. HASLETT FRAZER.
231, Macquarie Street,
Sydney,

April 18, 1946.

POLITICS AND THE MEDICAL PROFESSION.

SIR: It may, perhaps, be not too didactic on my part to suggest to Dr. Dark that although a verbal "expression" of thought by one person may appear to be "strange" to another, this word can hardly apply to "statements" made in regard to matters of extreme politico-economic importance, for such are merely true or false and thereby eventually pinning the correspondent down to a recognition of the correct meanings of the words thus being associated.

Property of any kind is either owned by the persons that have a legalized title thereto and the consequent right to use it to the best advantage—that is, subject to the ordinary control of government as in democracy—or, as in autocracy, it is owned and administered by the government class and which Dr. Dark confuses with "the whole community" in the same manner that the government class confuse themselves with "the State" and which latter is the whole community.

If "in a socialist State it is the whole community that owns the means of production, administered by its elected

representatives", will Dr. Dark give one illustration where these "elected representatives" administer their efforts at "production" in any way other than at a tragic loss and with uneconomic repercussions. If all people thought alike and acted in harmony and unity by nature his theories might deserve a clap of the hands. But "production" is a hefty word and likes to be in constant company with "distribution and exchange". There are only two systems of production—that is, barter and the capitalistic system—and, in the latter case, owes its comparatively high status to certain easily defined factors that together constitute "capital" on the part of the employers and employees respectively concerned in the primary, secondary and tertiary industries of the national supply, and the very king-pin of capital is essentially composed of such mental attainments as specialized knowledge, inventiveness, initiative, capacity to organize and general ability to conduct business in a profitable manner.

Dr. Dark asserts that "there are three absurdities" in my statements that "The word 'capitalism' implicates the capitalistic system of production, distribution and exchange, money and the gold standard. . . . The Soviet Union practises capitalism equally as the democracies", and would appear to imagine that these "absurdities" can be genuinely stamped as such on the grounds of his own three assertions (for I did not make them in my letter), namely: (*a*) "a capitalist State may or may not use the gold standard"; (*b*) "money is used by both capitalist and socialist states"; and (*c*) "the Soviet Union does not practise capitalism"; and which can be investigated *seriatim*.

In regard to (*a*) it may be stated that there exists no civilized State in the world that does not use the gold standard, for every transaction therein is completed by means of the domestic currency that has a rate of exchange with gold of fixed weight and quality and whether gold is legal tender or not within the country concerned. (*b*) If "money is used by both capitalist and socialist States", how could the latter avoid practising the capitalistic system? (*c*) If, again, "the Soviet Union does not practise capitalism", why does it use money and mine gold like fury?

Stalin's favourite industry is said to be the Gold Trust (see "In Search of Soviet Gold", by John D. Littlepage), and all the world wonders the amount of the Russian gold reserve.

"The fundamental difference between socialism and capitalism" is constituted as the comparative realities founded on which these two words are being irrefutably defined and clear reference was made thereto in my previous letter.

If, again, "socialists all wish to see democracy fulfilled", it would be interesting to hear Dr. Dark's philosophy as to what "democracy" signifies, for "equal opportunity to develop and use his abilities to the full" would appear to be eminently carried out in a system that permits the lowliest to, without force, rise to the highest positions of trust, wealth and power—that is, as definitely pertains in a democracy and provided they are endowed with sufficient idealism, constancy and character.

Dr. Dark would appear to merely beg the question with self-assumed infallibility when he prescribes "from each according to his ability, and to each according to his work", on the one hand, and, it seems, only starts his cure when "(*a*) that production is great enough to supply all the needs of every member of the community, (*b*) that the education of living in a socialist society has so developed the moral sense of the great majority of the people that they realize that they are 'members one of another'", on the other. Verily a "watch poppa and learn" proposition!

Yours, etc.,

138, Darlinghurst Road, Sydney,
April 16, 1946.

NORMAN PERN.

A DISCLAIMER.

SIR: Recently in one of the Sydney newspapers there appeared an appreciative article concerning the work of the Repatriation Department at the Lady Davidson Home, Turramurra.

Reference was made to a number of medical officers assisting at this sanatorium. I was quoted as a "Visiting Lung Specialist". This was done without approval or my knowledge.

Yours, etc.,

185, Macquarie Street, Sydney,
April 18, 1946.

A. W. MORROW.

INGUINAL HERNIA AND ITS REPAIR.

SIR: In a letter to THE MEDICAL JOURNAL OF AUSTRALIA, March 16, 1946, Lieutenant-Colonel L. S. Loewenthal reports that in a series of cases of hernia treated by him, 10% had both direct and indirect sacs. Would Lieutenant-Colonel Loewenthal kindly state whether it was possible to recognize this condition before operation?

Yours, etc.,

General Hospital, Launceston,
April 17, 1946.

C. CRAIG,
Surgeon Superintendent.

The Royal Australasian College of Surgeons.

GORDON CRAIG SCHOLARSHIPS.

THE Council of the Royal Australasian College of Surgeons invites applications for the second award of Gordon Craig Scholarships.

1. These scholarships are awarded for the purpose of furthering post-graduate education in surgery and surgical research.
2. Favourable consideration will be given to applicants who intend to present for a senior surgical qualification.
3. Applicants must have been qualified for at least two years.
4. Scholarship holders will be required to carry out whole-time duty at a medical school and/or research institute and/or hospital, although this condition may be varied in exceptional cases.
5. The amount of each scholarship will be at the discretion of the College, but the College is prepared to grant up to £400 a year to suitable applicants.
6. Forms of application, together with details of the regulations governing the award of the scholarships, are available from the Secretary, Royal Australasian College of Surgeons, Spring Street, Melbourne.
7. Applications should be lodged with the Secretary of the Royal Australasian College of Surgeons, Spring Street, Melbourne, and reach him not later than July 31, 1946.

It is proposed to call for further applications at approximately half-yearly intervals, and announcements will be made from time to time.

The Royal College of Obstetricians and Gynaecologists.

EXAMINATION FOR MEMBERSHIP.

A SPECIAL examination for membership of the Royal College of Obstetricians and Gynæcologists will be held in Sydney and Melbourne in February, 1947. When the plans of the Chief Examiner, Sir W. Fletcher Shaw, are known, the exact date will be notified to all candidates. The fee for the examination will be £25 sterling. Intending candidates who have not already forwarded their application forms should do so as soon as possible to Professor Marshall Allan, University of Melbourne.

Post-Graduate Work.

POST-GRADUATE WEEK IN BRISBANE.

THE following is the programme of post-graduate week which will be held at Brisbane from June 1 to 7, 1946. An announcement of the course appeared in last week's issue.

Saturday, June 1.

10 a.m.: Demonstration of treatment of fractures in the orthopædic departments of the Mater Misericordiæ and Brisbane General Hospitals.

2 p.m.: Medical School, Herston Road. A symposium on "Headache" from the (i) medical aspect (Dr. Ellis Murphy), (ii) surgical aspect (Dr. Konrad Hirschfeld),

(iii) psychosomatic aspect (Professor John Bostock), (iv) oto-rhino-laryngological aspect (Dr. Athol Quayle) and (v) ocular aspect (Major A. V. Henry).

Sunday, June 2.

9.30 a.m.: Anatomy Department, Medical School, Herston Road. Professor H. J. Wilkinson: "Main Stages in the Evolution of the Human Brain."

11 a.m.: Anatomy Department, Medical School, Herston Road. Dr. E. S. Meyers: Demonstration—"Fasciæ of Surgical Importance".

Monday, June 3.

9.30 a.m.: Brisbane General Hospital. Rounds, selected medical cases, Dr. Horace Johnson.

11.30 a.m.: Lecture Hall, Brisbane General Hospital. "Recent Advances in Drug Therapy", Dr. Otto Hirschfeld.

2.30 p.m.: Pathology Department, Medical School. Demonstration—museum specimens of pulmonary tuberculosis, Professor J. V. Duhig.

4.30 p.m.: Lecture Theatre, Medical School. "Modern Aspects of the Medical Treatment of Pulmonary Tuberculosis", Dr. C. H. Fitts. (Discussion to be opened by Dr. W. H. Steele.)

8 p.m.: Lecture Theatre, Medical School. "Surgical Aspects of the Treatment of Pulmonary Tuberculosis", Mr. C. J. Officer Brown. (Discussion to be opened by Dr. Alan Lee.)

Tuesday, June 4.

9.30 a.m.: Gymnasium, Medical School, Herston Road. Demonstration in the use of physiotherapy in chest surgery by Dr. Harold Crawford and the staff of the Physiotherapy Department.

11.30 a.m.: Lecture Theatre, Medical School. "The Diagnosis of Fevers", by Dr. E. H. Derrick.

2 p.m.: Operating Theatre, Brisbane General Hospital, Dr. G. H. Brandis.

4.30 p.m.: Lecture Hall, Brisbane General Hospital. "The Scope of Thoracic Surgery in Non-Tuberculous Conditions", Dr. C. J. Officer Brown. (Discussion to be opened by Professor Neville Sutton.)

8 p.m.: Lecture Theatre, Medical School, Herston Road. "Cough as a Symptom, with Some Remarks on Treatment", by Dr. C. H. Fitts. (Discussion to be opened by Dr. S. F. McDonald.)

Wednesday, June 5.

9.30 a.m.: Brisbane General Hospital. Rounds, selected medical cases, Dr. Ellis Murphy.

11.30 a.m.: Lecture Theatre, Brisbane General Hospital. "Vascular Disturbances of the Nervous System", Dr. Ellis Murphy.

Afternoon: Conference of the delegates of the local associations of the Queensland Branch of the British Medical Association.

Evening: British Medical Association dance, Lennon's Hotel.

Thursday, June 6.

9.30 a.m.: Brisbane Women's Hospital, Bowen Bridge Road. Technique of the investigation and treatment of sterility, Professor G. Shedden Adam. (Class limited to ten members.)

12 noon: Lecture Hall, Brisbane General Hospital. Screening by Wyeths of "Peptic Ulcer" film from the Lahey Clinic.

2.30 p.m.: Mater Misericordiæ Public Hospital. Rounds, selected medical cases, Dr. Norman Sherwood.

4.30 p.m.: Lecture Hall, Mater Misericordiæ Public Hospital. "The Diarrhœas", by Dr. Norman Sherwood.

8 p.m.: The Sir William Macgregor School of Physiology, William Street (behind the Belle Vue Hotel). "Some Impressions of Medical Science Overseas", Professor D. H. K. Lee.

Friday, June 7.

9.30 a.m.: Brisbane Children's Hospital, Herston Road. Rounds, selected medical cases, Dr. S. F. McDonald.

11.30 a.m.: Brisbane Children's Hospital, Herston Road. Demonstration of intravenous infusions and blood transfusion in infancy and childhood, Dr. Felix Arden.

2.30 p.m.: Mater Misericordiæ Children's Hospital. Rounds, selected medical cases, Dr. P. A. Earnshaw.

4 p.m.: Lecture Hall, Mater Misericordiæ Public Hospital. "Convulsions in Infancy and Childhood", Dr. P. A. Earnshaw.

8 p.m.: Lecture Theatre, Medical School, Herston Road. Bancroft Memorial Lecture, 1946. Dr. A. Holmes à Court: "Functional Disorders Sequent to Organic Disease."

Members of the course are advised that luncheon may be obtained at the café attached to the Victoria Park Golf Club opposite the Hospital for Sick Children, Herston Road.

COURSE ON HEART DISEASE AT ADELAIDE.

THE Post-Graduate Committee in Medicine of the University of Adelaide announces that a week-end course on the subject of heart disease will be held in the Verco Lecture Theatre, at the Institute of Medical and Veterinary Science, Frome Road, Adelaide, on Saturday and Sunday, June 15 and 16, 1946, provided there are at least six applicants for attendance. A similar course on a surgical subject will be held in August and a third course will be held in October on obstetrical subjects.

Applications close on Saturday, June 1, 1946, and should be sent to the Medical Secretary, c.o. the University of Adelaide, North Terrace, Adelaide. Applicants should register with Dr. J. M. Bonnin, the Medical Secretary, in Room 121 of the Institute of Medical and Veterinary Science, Frome Road, Adelaide, between the hours of 12 noon and 2 p.m. on Saturday, June 15, 1946. The fee for the course is £3 3s., which may be sent by cheque with the application or paid in person to the Medical Secretary on registration.

The programme is as follows:

Saturday, June 15.

2-3 p.m.: Anatomy of the heart, Professor Abbie.

3.15-4 p.m.: Coronary artery disease, Dr. E. F. Gartrell.

4.30-5.30 p.m.: Treatment of congestive cardiac failure, Dr. G. A. Lendon.

8-9.30 p.m.: Pathology, Dr. J. B. Thiersch.

Sunday, June 16.

10-11 a.m.: Physiology, Sir Stanton Hicks.

11.15 a.m.-12.15 p.m.: Subacute bacterial endocarditis, Sir Trent de Crespigny.

2.30-4 p.m.: Examination of patients at Estcourt House,[1] Dr. E. Britten Jones.

[1] This item will be included only if sufficient applicants wish to attend. Applicants should state whether they desire to attend at Estcourt House when sending in their applications.

A FILM AFTERNOON AT SYDNEY.

THE New South Wales Post-Graduate Committee in Medicine announces that the following films will be shown in the Stawell Hall, 145, Macquarie Street, Sydney, at 4.15 o'clock p.m. on Wednesday, May 22, 1946: "Nutritional Diseases"; "An Operation for Hydatid of the Liver"; "The Perineo-Abdominal Resection of the Rectum"; "Fractures of the Neck of the Femur". All medical practitioners are invited to be present.

Australian Medical Board Proceedings.

QUEENSLAND.

THE undermentioned have been registered, pursuant to the provisions of *The Medical Acts*, 1939, of Queensland, as duly qualified medical practitioners:

Ahern, Thomas Francis, M.B., 1940 (Univ. Sydney), 58, Ascot Terrace, Toowong, Brisbane.

Ley, Thomas Urban, M.B., B.S., 1922 (Univ. Melbourne), M.Ch.Orth., 1937 (Univ. Liverpool), Townsville.

McDowell, Moira Agnes, M.B., 1945 (Univ. Sydney), General Hospital, Brisbane.

Robertson, Athol Herbert, M.B., B.S., 1938 (Univ. Melbourne), D.T.M. and D.T.H., 1944 (Univ. Sydney), Brisbane Clinic, Wickham Terrace, Brisbane.

Flecker, Patrick Oscar, M.B., B.S., 1944 (Univ. Adelaide), District Hospital, Mareeba.

Nominations and Elections.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Stanton-Cook, Peter Alan, M.B., B.S., 1945 (Univ. Sydney), 9, Coolawin Road, Northbridge.

Frost, Thomas James, M.B., B.S., 1945 (Univ. Sydney), 28, Fern Street, Randwick.

Courtenay, Kevin Hector, M.B., B.S., 1945 (Univ. Sydney), 33, William Street, Double Bay.

Grunseit, Ferry, M.B., B.S., 1945 (Univ. Sydney), 17, Miller Road, Old Guildford, New South Wales.

Yuille, Archibald Binnie, M.B., B.S., 1942 (Univ. Sydney), Barrenjoey Road, Newport Beach, New South Wales.
Filshie, Ian, M.B., B.S., 1941 (Univ. Sydney), 10, Herbert Street, Manly.
De Salis, Eric James Fane, M.B., 1942 (Univ. Sydney), 101 Australian General Hospital, Punchbowl.
Farrar, Frank Martindale, junior, M.B., B.S., 1945 (Univ. Sydney), Sydney Hospital, Macquarie Street, Sydney.
Hunt, Thelma May, M.B., B.S., 1945 (Univ. Sydney), 716, New Canterbury Road, Hurlstone Park.

THE FEDERAL MEDICAL WAR RELIEF FUND.

THE following contributions to the Federal Medical War Relief Fund have been received:

South Australia.

T. G. Wilson, £50.
C. O. F. Rieger, T. L. McLarty, M. Erichsen, £10 10s.
F. St. J. Poole, D. Macdonald Steele, £10.
D. T. M. Hayes, W. Gilfillan, P. F. Stratmann, H. M. Jay, Ruth Heighway, £5 5s.
R. S. Wilkinson, £3 3s. 3d.
P. W. Verco, R. R. Sobey, £3 3s.
M. G. Gratton, £2 2s.
R. D. Hammill, £2.
C. S. Swan, £1 1s.
Total: £142 7s. 3d.

Queensland.

W. W. Hoare, Walter Crosse, £50.
R. J. Nash, E. W. Casey, J. G. Wagner, Isabel McLelland, Roy Hemsley, Doris U. Skyring, Anonymous, £10 10s.
Val. McDowall, J. A. Spalding, Evan R. W. Thomson, £10.
H. W. Horn (first donation), E. R. Watkins, £5 5s.
Elizabeth Coles, F. W. R. Lukin, H. N. Merrington, S. V. O'Regan, £5.
Elizabeth Goldberger, Donald A. Cameron, £3 3s.
Jean S. Stobo, J. H. Blackburn, A. J. Buchanan, K. B. Shallard, J. R. Robertson, R. L. Rankin, £2 2s.
Anonymous, G. E. B. Clayton, £1 1s.
L. Comino, £1.
Total: £256.
Grand total: £7,065 7s. 6d.

Books Received.

"Physical Chemistry of Cells and Tissues", by Rudolf Höber, with the collaboration of David I. Hitchcock, J. B. Bateman, David R. Goddard and Wallace O. Fenn; 1945. London: J. and A. Churchill Limited. 9¼" × 6", pp. 709, with many illustrations. Price: 42s.

"Medical Jurisprudence", by W. F. Rhodes, B.A., M.B., Ch.B. (Cantab.), D.P.H. (Oxon.), I. Gordon, M.B., Ch.B. (Cape Town), and R. Turner, M.B., Ch.B., D.P.H. (Cape Town), with additional contributions by M. R. Drennan, G. W. Gale, J. A. Keen, G. J. Key and H. A. Shapiro; foreword by the Honourable Mr. Justice H. S. Van Zijl; Second Edition; 1945. Cape Town: The Post-Graduate Press. 8½" × 5½", with many illustrations. Price: 32s. 6d.

"The Problem of Lupus Vulgaris", by Robert Aitken, M.D., F.R.C.P.E., F.R.S.E.; 1946. Edinburgh: E. and S. Livingstone Limited. 8½" × 5½", pp. 78, with many illustrations, some of them coloured. Price: 15s. net, postage 7d.

"The 1945 Year Book of Obstetrics and Gynecology", edited by J. P. Greenhill, B.S., M.D., F.A.C.S.; 1946. Chicago: The Year Book Publishers Incorporated. 7" × 4½", pp. 576, with illustrations. Price: 24s.

Medical Appointments.

Dr. T. A. Daly has been appointed Government Medical Officer at Parramatta, New South Wales.

Dr. Michael Slavin has been appointed Medical Officer of Health, Northampton Road Board, Western Australia.

Dr. F. L. Gill has been appointed a member of the Medical Register Committee under section 23 of the *Workers' Compensation Act*, 1912-1941 (as reprinted pursuant to the *Amendments Incorporation Act*, 1938), Western Australia.

Dr. Dion Gavan Flanagan has been appointed deputy quarantine officer at Melbourne, Victoria, in accordance with the provisions of the *Quarantine Act*, 1908-1924.

Dr. Victor Henry Webster, Dr. Edgar Herz and Dr. Harry Campbell Pope have been appointed quarantine officers respectively at Wyndham, Derby and Port Hedland, Western Australia, under the provisions of the *Quarantine Act*, 1908-1924.

Diary for the Month.

MAY 14.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
MAY 14.—Tasmanian Branch, B.M.A.: Ordinary Meeting.
MAY 15.—Western Australian Branch, B.M.A.: General Meeting.
MAY 16.—Victorian Branch, B.M.A.: Executive Meeting.
MAY 16.—South Australian Branch, B.M.A.: Council Meeting.
MAY 21.—New South Wales Branch, B.M.A.: Medical Politics Committee.
MAY 22.—Victorian Branch, B.M.A.: Council Meeting.
MAY 23.—New South Wales Branch, B.M.A.: Clinical Meeting.
MAY 24.—Queensland Branch, B.M.A.: Council Meeting.
MAY 28.—New South Wales Branch, B.M.A.: Ethics Committee.
MAY 30.—South Australian Branch, B.M.A.: Council Meeting.
MAY 30.—New South Wales Branch, B.M.A.: Branch Meeting.
JUNE 4.—New South Wales Branch, B.M.A.: Organization and Science Committee.
JUNE 5.—Victorian Branch, B.M.A.: Branch Meeting.
JUNE 5.—Western Australian Branch, B.M.A.: Council Meeting.
JUNE 6.—New South Wales Branch, B.M.A.: Special Groups Committee.
JUNE 7.—Queensland Branch, B.M.A.: Branch Meeting (Bancroft Memorial Lecture).

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Honorary Secretary, 135, Macquarie Street, Sydney): Australian Natives' Association; Ashfield and District United Friendly Societies' Dispensary; Balmain United Friendly Societies' Dispensary; Leichhardt and Petersham United Friendly Societies' Dispensary; Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney; North Sydney Friendly Societies' Dispensary Limited; People's Prudential Assurance Company Limited; Phœnix Mutual Provident Society.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federated Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178, North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

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